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ORIGINAL LECTURES.

CLINICAL LECTURE ON PYÆMIA.

Delivered at the College of Physicians and Surgeons, Chicago, Illinois,

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GENTLEMEN,—We shall consider this morning what remains of the long list of wound infective diseases,—pyæmia. With Gussenbauer, I will define pyæmia as a general infective disease, the result of the introduction into the blood of the products of septic pus; differing from other infectious diseases by the presence of multiple foci of inflammation and suppuration, and attended by an intermittent form of fever. The term pyæmia was introduced by Piorry, and literally signifies simply the coexistence of blood and pus,—*a pyo-hæmia.* Even before the relations of suppuration to pyæmia were known, it was supposed that it was the direct result of introduction into the circulation of certain noxious fluid substances (the humoral doctrine of pyæmia). Piorry recognized the direct relation between suppuration and pyæmia, and believed that the disease was due to the direct introduction of pus into the circulation. You must remember that at that time the histology of pus was very little understood; it was supposed to be simply a fluid which might permeate the vascular wall, and which, by deposition in certain regions of the body, was supposed to produce secondary or metastatic abscesses. This was the early pathological view of pyæmia,—the direct entrance of pus from the local point of suppuration into the circulation, and its arrest and localization within certain vessels in distant parts of the body.

Then came the view of Hunter, who believed that pyæmia was always the result of phlebitis; that the inflammation of the vein-wall, by producing pus, was the cause of secondary or metastatic abscesses (the phlebitic theory of pyæmia). He believed that from the tissue of the vein-walls (the seat of the suppurative inflammation) the inflammatory products gained direct access into the interior of the inflamed vessel, and that by a process of filtration, or in consequence of an arrest of the circulating pus in the blood, the numerous metastatic abscesses were produced.

Cruveilhier made the assertion that in all instances of phlebitis thrombosis was the primary cause of the inflammation; that the peculiar characteristic features of pyæmia consisted in a primary thrombosis; in short, there was, first, the formation of a thrombus in a vessel, and, secondly, a subsequent or secondary inflammation in the immediate vicinity of the thrombus. This idea was carried so far by his disciples that all inflammatory processes were by them attributed to thrombotic changes in the smallest veins: not only inflammatory lesions, but even new growths, tumors, tuberculosis, etc., were supposed to originate in small veins by the initiation of a local inflammation resulting in various pathological products. A new aspect was given to the pathology of the disease by the careful experimental investigations of Virchow on thrombosis and embolism. He showed that the metastatic deposits always occurred at points where vessels had been blocked by an embolus derived from a disintegrating thrombus. While the direct relationship existing between suppuration and pyæmia was well understood clinically, it was left for Klebs to demonstrate for the first time the direct connection of the pyæmic processes with the presence of specific microbes. In his researches into the nature of the disease during the Franco-Prussian war in 1870, he detected in the pyæmic products certain well-defined morphological elements which he called the *micrococci of pyæmia.* He found that these micro-organisms always arranged themselves in the form of "colonies" or "groups," which he termed "zoöglœa." He found this specific germ invariably present, notably at the primary seat of infection, but also in the most distant organs,—wherever, indeed, pathological changes during the course of the disease occurred.

Pasteur, in studying the puerperal form of pyæmia, detected the same morphological elements, but he found that they arranged themselves in the form of chains, consequently he called them the *microbe en chapelet,* or "chain coccus."

Vogt, when studying this subject under

the direction of Hueter, found certain germs which he called "monads" always present in all pyæmic deposits.

Burdon-Sanderson supposed that he had detected the essential cause of pyæmia in a "dumb-bell-shaped germ." From our present stand-point we can readily assume that the shape of the germ described by Sanderson was determined by the approximation of two micrococci, which when brought into close contact would resemble a dumb-bell.

The first reliable investigation into the nature of the disease was made by Koch, who not only studied the form and natural history of these germs, but produced pyæmia artificially. He found that by obtaining a putrid fluid by maceration of the skin of a mouse in distilled water, and injecting a considerable quantity of this fluid into the subcutaneous tissue of a rabbit, it induced a rapidly fatal form of pyæmia. He found that after the animal had died,—five hours to five days after inoculation,—at the point of injection there was an extensive subcutaneous abscess; and on pursuing his investigations further, he found in the various distant organs characteristic pyæmic metastatic deposits, capillary metastases, and multiple foci of inflammation and suppuration, terminating in secondary inflammations in adjacent organs, as, for instance, suppurative pleuritis the result of metastatic abscesses underneath the pleura, and suppurative peritonitis from similar processes beneath the peritoneum. By taking the serum from the œdematous portion of the pathological product and injecting it again into another animal it produced the same conditions, but in a much shorter time. He ascertained also that by diminishing the dose the interval between inoculation and the fatal termination was invariably increased, showing conclusively that in regard to time the pathological effect of the specific cause of pyæmia holds a direct relationship to the number of germs introduced. He described these germs as the micrococci of pyæmia, which in appearance resemble closely the zoöglœa described by Klebs.

Rosenbach went one step further, and examined the pyæmic deposits in man, and by cultivating directly upon an artificial soil properly sterilized he obtained the specific germs of the disease. In five out of six cases he found the *streptococcus pyogenes;* he found the *chain coccus* and the *staphylococcus* in combination in two of the five cases; and in one case only of pyæmia he found the staphylococcus alone, and this was the only case that recovered. These observations show conclusively that in the majority of cases the germs which produce the disease are the ordinary germs of suppuration first described by Ogston. Subsequent experience has shown that when the disease is produced by the staphylococcus alone it usually assumes a milder type. The results of these experimental and clinical investigations demonstrate conclusively the essential etiological connection that exists between suppurative lesions and pyæmia,—viz., that in every case suppuration must precede as a necessary antecedent pathological condition, in contradistinction to septicæmia, as I have informed you on a previous occasion, where the existence of suppuration is irrelevant: septicæmia simply being a disease due to putrefaction and absorption of wound-products, whilst pyæmia is invariably the result of an infection from a dépôt of suppuration.

We shall take it for granted that in every case of pyæmia we are dealing with the presence of the essential germs (the streptococcus and the staphylococcus), which produce at first a local suppuration, and secondly, when the disease becomes general, implicate the veins. This leads us to a consideration of phlebitis and the etiological relationship which exists between phlebitis and thrombosis on the one hand, and between phlebitis and suppuration on the other. The etiologico-clinical feature of a pus-microbe in a pyæmic patient consists in its capacity to permeate the vascular wall. The route which the specific germ of the disease selects in effecting entrance into the circulation is always through a vein or capillary vessel. As in the production of a suppurative inflammation in the soft tissues anywhere, the specific primary effect of the germ is upon the tissues with which it first comes in contact; so in cases of pyæmia the contact of the germ with the vessel-wall produces a destructive parenchymatous phlebitis. Whilst we recognize the truth of Virchow's assertion that a simple or plastic phlebitis can run its course without producing thrombosis, it is different in cases of pyæmia, as the phlebitis associated with this disease is always of an infective type and assumes a necrotic character: in other words, the contact of the specific germs

with the living vessel-wall produces a coagulation-necrosis or interstitial death of the tissues, and is consequently always followed, as a necessary consecutive result, by thrombosis *in loco* at the primary point of inoculation. We have here a pathological condition as we observe it in man, as compared with the direct and rapid results produced by Koch upon animals by the direct introduction of the pyogenic agents into the subcutaneous tissue. Clinically, we observe that the inoculation of the tissues in a wound with pus-microbes in the immediate proximity of a vein is followed by a primary pyogenic phlebitis, and, as a result of microbic action, death of the endothelial lining ensues, and the consecutive coagulation-necrosis leads to conglutination and coagulation and the formation of a thrombus. This thrombus subsequently is infiltrated with the specific germs, which again facilitate or hasten necrotic changes in the thrombus, which results in puriform softening and disintegration. Phlebitis in connection with pyæmia signifies always the production of a thrombus at the point of inflammation, and its subsequent dissemination by embolism. The *infection-atrium*, or route by which the pus-microbes gain entrance into the system, is usually through a wound,—a loss of surface-continuity somewhere which may not be detected or recognized in every instance, as in some cases of septicæmia, for the primary wound may have healed before pyæmia has occurred. But, as it is quite rational to assume that the germs of septicæmia enter the circulation only through an abraded surface, so in pyæmia the actual development of the disease is always preceded by some form of traumatism or some other appreciable antecedent pathological condition. A form of pyæmia has been recently described where no initiatory lesion can be found, and which has been referred to so-called auto-infection, which we can only explain by the entrance of the specific germs into the circulation through intact mucous surfaces and their localization in some internal organ, where they remain in a latent condition for an indefinite period of time, but sooner or later, under the influence of some determining cause or causes, produce local pathological conditions at the seat of deposition which lead to suppuration, thrombo-phlebitis, and pyæmia.

In considering the symptoms we must first allude to the local conditions in or about the wound. In patients threatened with pyæmia we usually observe a well-defined train of premonitory symptoms which precede the actual development of the disease, referable to the appearance of the wound and the general condition of the patient. The existence of the disease may be suspected at any time after suppuration has occurred, when evidences of serious capillary stasis manifest themselves at the seat of injury or operation. As the result of this intense capillary congestion at the primary seat of infection we observe a localized œdema; the margins of the wound appear puffed and elevated; the granulations pale and flabby; suppuration, which may have been profuse, becomes scanty; the pus changes its character, and, instead of a yellowish cream-colored fluid, it becomes sanious, serous, or sero-sanguinolent. On close inspection, in many instances, you are enabled at this time to detect direct evidences of thrombosis in some of the larger veins, manifested by all the symptoms resulting from venous obstruction. The general premonitory symptoms indicate a slight degree of intoxication, the result of the introduction of ptomaines from the primary suppurative dépôt into the circulation,—a general feeling of malaise; loss of appetite; sometimes a slight increase of temperature, and a slight acceleration of the pulse. The increase of temperature is followed sooner or later by the actual development of the disease, which is always initiated by a sudden, well-defined chill, which lasts from a few minutes to an hour or more. A chill of this kind must always be considered a grave symptom, as it indicates that a large amount of ptomaines has been suddenly introduced into the circulation. The chill usually recurs at irregular intervals,—one, two, or three a day, as a rule,—increasing in frequency, and often in intensity, as the disease progresses. If, for instance, during the first few days your patient has one daily chill, and, after a few days, two or three chills a day, every additional chill indicates a more advanced degree of intoxication and a wider diffusion of the ptomaines throughout the organism. New foci of infection near and remote from the primary seat of infection serve as independent centres for the production of ptomaines; consequently the

greater the area of infection the more grave the condition of the patient, and the greater the intensity of the general symptoms. The pyæmic chill inaugurates an intermittent type of fever, which in many instances resembles malarial fever almost to perfection; in fact, many cases of pyæmia have been mistaken in the beginning for cases of malarial fever, and *vice versa;* but, instead of the regular periodical chills such as characterize malaria, no regular periodicity is observed in pyæmia. You rarely, if ever, have two daily chills in malaria; but two or three or four chills in twenty-four hours are not infrequent in cases of pyæmia. As in malarial fever, so in pyæmia, the thermometer shows a rise of temperature during the algid stage, which rapidly increases, and reaches its maximum before the sweating stage. The temperature may become subnormal during the remissions or intermissions, showing that the chilly sensations are entirely subjective, as at this time the thermometer invariably indicates a constant rise in temperature. The circulatory organs, as in cases of sepsis, are deleteriously affected by the ptomaines, which act as direct cardiac sedatives. You observe the same feeble, rapid pulse, but the depression is not as marked as in most cases of acute septicæmia, as pyæmia is characterized by a slower process, a more gradual development of symptoms, and an inherent tendency to localization. The respiratory organs furnish additional and important symptoms, which, when present, are often sufficient upon which to base a differential diagnosis between this disease and septicæmia, as we shall see farther on. The secondary changes characteristic of pyæmia almost invariably occur first in the lungs; consequently we may expect that the local foci of inflammation in this organ will be attended by a change of function, as manifested by rapid respiration, etc. The clinical evidences which indicate well-defined points of localization of these secondary processes, such as consolidation of certain areas of lung-tissue each surrounded by a zone of local hyperæmia and œdema, and caused by the metastatic deposits, will be discovered at the bedside by physical exploration of the chest, which yields localized râles, increased bronchial sounds over the solidified lung, and all the evidences of diffuse multiple foci of inflammation. As in cases of septicæmia, so in pyæmia, the gastro-intestinal canal is always involved in the morbid process, as the mucous membrane invariably shows evidences of capillary stasis, œdema, or even catarrhal inflammation, which accounts for the total loss of appetite, vomiting, and diarrhœa, which are frequent and conspicuous symptoms. The nervous symptoms in pyæmia differ from those of septicæmia, inasmuch as they are not so marked; and when they are prominent they simply indicate, as in septicæmia, a rapid production and diffusion of ptomaines, which produce a sedative effect directly upon the brain and nervous system in general.

In considering the differential diagnosis you must also remember the subject of fermentation fever, which, when it occurs after an injury or operation, always appears within twenty-four hours, and is caused by absorption of fibrin ferments, and not by infection; while pyæmia never occurs before sufficient time has elapsed for suppuration to become established, which always requires at least three or four days, while it may occur at any subsequent time, so long as suppuration continues. Remember that suppuration bears a direct etiological bearing to pyæmia, and always precedes and attends this disease. Septicæmia you will recognize by the most prominent symptoms, such as a slight chill, usually not repeated, the initiatory sensation of chilliness being followed by a continuous form of fever, in contradistinction to the repeated chills and the intermittent form of fever attending pyæmia. It is sometimes more difficult to differentiate between polyarthritis of a non-pyæmic character and true pyæmia. For instance, you may mistake, possibly, an acute articular rheumatism for pyæmia, and *vice versa*, inasmuch as the disease under consideration frequently affects multiple joints, producing multiple suppurative arthritis; but you must recollect that rheumatic fever follows a well-defined course, with a definite history and a continuous form of fever, while, on account of the progressive infection, pyæmia is attended by an intermittent form of fever. Then, again, carefully consider the clinical history of the case, with special reference to the existence of a dépôt of suppuration caused by the presence of antecedent pathological suppurative products within the body caused by auto-infection.

If any doubt remains as to the nature of the local inflammation in the joint or joints, an exploratory puncture will reveal the character of the pathological product, and thus render valuable diagnostic assistance.

In cases of gonorrhœal arthritis the question of differential diagnosis becomes still more difficult, inasmuch as this form of arthritis has been considered by a number of eminent pathologists as a mild form of pyæmia, an opinion which now appears substantiated upon a pathological basis, as suppuration of the urethral mucous membrane always precedes the development of the disease; consequently the difficulty of diagnosis is diminished if you are able to trace the disease to its specific source.

The morbid anatomy of pyæmia furnishes an interesting and important subject for discussion. First, let us consider the primary focus of suppuration, caused by the direct introduction of the microbes of pus, which produce the local suppuration. The question naturally presents itself, Why do not all suppurative processes lead to pyæmia? Theoretically, an explanation can be advanced by assuming that in some forms of suppuration the pus-microbes possess a less degree of potency to infect the tissues, or else that the organism or tissues of the patient present a greater degree of resistance. Those conditions which would tend to limitation of the disease, and thus calculated to protect the patient against pyæmia, must be carefully considered in studying the causative relations between suppuration and pyæmia. The anatomical structure at the seat of the primary suppuration also exerts a potent influence in determining the occurrence of the disease; for it is a well-known clinical fact that suppurative osteo-myelitis is frequently followed by pyæmia, while suppuration in other localities, even when very extensive, is seldom followed by the extension of the suppurative process to veins. The actual development of the disease begins with a thrombo-phlebitis at the primary suppurative dépôt, from which the subsequent embolic infarcts in distant organs are derived by disintegration of the thrombus and transportation of fragments to the nearest filtrum, the lungs. The primary phlebitis is due to the pus-microbes having come in contact with and having infiltrated the vein-wall, and produced tissue-changes which favor coagulation-necrosis, consequently the formation of a thrombus *in loco*. The phlebitis at the seat of embolism is caused by the transportation of a fragment of the primary thrombus or the implantation of the specific germs upon the intima of distant vessels, producing an arteritis from within outward, in contradistinction to phlebitis from without inward, as was the case at the primary dépôt of the pyæmic process. It has been shown experimentally that certain chemical irritants, when applied to the exterior of a vein, produce a direct necrotic effect upon the intima without necessarily producing a parenchymatous inflammation of the intervening tunic. For instance, the application of nitrate of silver to the jugular vein of dogs produces a necrosis of the intima, while the remaining portion of the vein-wall remains intact. We must assume that the nitrate of silver reaches the intima by diffusion where death of the endothelial cells is produced by the chemical action upon their protoplasm. It is reasonable to assign to the action of the ptomaines a similar destructive effect upon the tissues with which they come in contact, either directly by the medium of the germs, or by diffusion. It has also been demonstrated that *digitoxin*, a vegetable substance, when brought in contact with endothelial cells by intravenous injection, produces necrosis of the intima. From these experiments it appears more than probable that the ptomaines elaborated by the pus-microbes, and brought in contact with the intima, produce similar necrotic effects, which determine the formation of a thrombus *in loco* by conglutination and coagulation in all cases where a secondary thrombus forms independently of an embolic infarct. The primary thrombo-phlebitis is doubtless produced by the deleterious effects of the pus-microbes, the ptomaines acting upon the vessel-wall in a centripetal direction, while the thrombus at the seat of metastasis is produced by an embolic infarct or the implantation of the essential germs upon the intima, where they or their product, the ptomaines, destroy the vitality of the intima. This condition is necessarily followed by coagulation-necrosis and the formation of a septic thrombus and extension of the inflammatory process in a centrifugal direction.

We have now reached the important subject of thrombosis from pathological causes. I have described to you in a pre-

vious lecture the process of coagulation as we observe it in vessels obstructed by the application of a ligature completely and suddenly interrupting the circulation, and detailed to you the minute processes which cause the formation of a non-infective or plastic thrombus. In such instances the intravascular coagulum results from the circumscribed tissue-changes in the vessel-wall following the trauma and the sudden and complete arrest of the circulation, conditions which induce coagulation-necrosis, the liberation of fibrin-ferment, and which, acting upon the remaining fibrin-producing substances (fibrinogen and fibrino-plastic material), causes the formation of a thrombus. Such a thrombus, due to aseptic causes, soon becomes infiltrated with the products of tissue-proliferation from the vessel-wall, and having performed its temporary function as a nidus for the plastic material, is completely removed by substitution on the part of the intra-vascular granulation-tissue, which by cicatricial contraction effects the definitive closure of the lumen of the vessel. Pathologists have been for years endeavoring to find a tangible explanation of the formation of the white mural thrombus during life which has been so often found upon post-mortem examination, and for the occurrence of which no satisfactory explanation could be given. The older pathologists interpreted the existence of a white mural thrombus by considering it as a remnant of an antecedent red or coagulation thrombus, decolorization having taken place by escape or destruction of the red corpuscles. Recent investigations, more especially the researches of Eberth and Schimmelpfennig, have shown that the white thrombus within a vessel during life has a different significance, as far as its production and duration are concerned. Such a thrombus is not caused by coagulation-necrosis alone; it consists almost exclusively of the third corpuscles of the blood, which become deposited by conglutination upon a projecting devitalized surface of the intima.

The interesting and valuable experiments of Eberth, for instance, have demonstrated that whenever a foreign substance encroaches upon the axial current of the circulation it mechanically arrests the third corpuscles which come in contact with it, a process which is termed conglutination, and which results in the formation of a white thrombus. If a thread be passed through an artery or vein of a living animal, it becomes the nucleus for a white thrombus which is composed almost exclusively of the third corpuscles. It has been claimed that these minute disks were the result of disintegration of pre-existing red or white corpuscles, the product of a retrograde change. But careful physiological investigation has verified the existence of a third element in the blood, whose function so far has not been definitely determined, but which, as we have seen, constitutes the most important element in the formation of a mural thrombus and the gradual obliteration of the vessel.

Another important element in the production of a thrombus in an inflamed vessel is the white corpuscle, which under such circumstances, in virtue of an increased viscosity, manifests a strong tendency to mural implantation, which, when the vessel is already the seat of mural thrombosis, accelerates the growth of the thrombus, and consequently the obliteration of the vessel. In capillary vessels, the seat of localization of the pus-microbes, rapid and complete stasis takes place by mural implantation of the viscous white corpuscles, which arrest the axial current, producing capillary thrombosis.

I hope I have made this subject sufficiently intelligible for you to appreciate the process of thrombosis as we observe it in cases of pyæmia. In recapitulation, it can be said (1) that the thrombo-phlebitis at the point of primary infection is caused by the action of the pus-microbes on the vessel-wall, causing tissue-changes which determine thrombosis. (2) The thrombus thus produced is an infective one, incapable of removal by granulation-tissue, destined to undergo disintegration, and by detachment of fragments it gives rise to embolism. (3) When pus-microbes have gained entrance into the venous circulation, they may produce thrombo-arteritis in distant parts by implantation upon the intima, where they give rise to necrobiotic changes which determine thrombosis and extension of suppurative inflammation to the remaining tunics and the paravascular tissues, which leads to the formation of metastatic abscesses.

On examining a recent thrombus in a case of pyæmia, you will always observe the zoöglœa in its peripheral portion show-

ing distinctly the etiological relations which they bear to it. When a sufficient period of time has elapsed for the germs to infiltrate the thrombus, they impart to all the elements of that thrombus the infective properties which characterize the primary dépôt of infection. The infection becomes general by dissemination of the pus-microbes set free by the thrombus and diffusion of the ptomaines through the circulation, these giving rise to the toxæmia which constitutes one of the most conspicuous clinical features of pyæmia. The infective germs which enter the veins at the primary infection-atrium reach the lungs as the first filtrum through the intervening venous channels directly, or through the medium of the white corpuscles or detached fragments of a disintegrated thrombus, where they initiate a septic thrombo-arteritis which constitutes the nucleus or starting-point for the formation of metastatic abscesses in this organ. In case the infective germs or fragments of a thrombus pass the first filtrum, they enter the left side of the heart, and by dissemination through the systemic circulation lead to metastatic abscesses in the most distant organs in the same manner as in the lungs. One of the characteristic features of pyæmia aside from the demonstrable embolism and metastatic abscesses is the existence of extensive capillary stasis, caused by direct implantation upon the lining of the capillary wall of floating germs, which so alter the tissues as to determine mural aggregation of the white corpuscles and complete closure of the lumen of the vessels by conglutination of the third corpuscle, conditions which finally result in the formation of a capillary thrombus.

The question, important to pathologists, as to the true relationship of phlebitis to thrombosis, has recently been satisfactorily explained. If thrombosis follow a non-infective plastic phlebitis, we shall find the thrombus surrounded by the inflamed vascular tunics, with a beginning infiltration of its periphery by the products of cell-proliferation from the fixed tissue-cells, as well as by migration corpuscles. The appearance of the vascular walls, as well as the condition of the thrombus, indicates that the inflammatory processes preceded the formation of the thrombus, and consequently must be considered as the direct cause of the intravascular coagulation-necrosis. If the reverse be the case, —if the thrombus be the cause of the phlebitis, as in cases of thrombosis after embolism, or after complete obstruction in a vessel from other causes,—on examination it will be found that the thrombus has undergone changes which antedated the inflammatory changes in the tunics of the vessels, and therefore must be looked upon as the direct cause of the phlebitis.

Having described thrombosis as it occurs in pyæmia at the primary point of infection and in distant organs, I will now call your attention to embolism. You must bear in mind that a primary thrombus is always caused by local causes in the vessel; its growth is gradual; consequently it does not give rise to sudden obliteration of a vessel, and in the majority of cases when due to pathological causes it is found in veins. Embolism, on the other hand, implies the sudden obliteration of a vessel, always an artery, by the impaction of the lumen of the vessel by a foreign substance or pathological product, which is carried in the direction of the blood-current until its further progress is mechanically arrested, and in consequence the circulation is suddenly and completely interrupted beyond the seat of the obstruction, unless subsequently restored by collateral circulation. Both of these forms of vessel-obliteration are met with in pyæmia. A plastic thrombus, as we have already seen, is capable of undergoing complete absorption by a process of substitution on part of the granulation-tissue springing from the fixed tissue-cells of the vessel-tunics; but an infected thrombus, the seat of infection of pus-microbes, is not capable of undergoing such favorable retrograde changes. This variety of thrombus disintegrates at an early stage by gradual granular degeneration of its component parts under the influence and by the cause of these specific microbes. There is no absorption, but a softening takes place primarily in the centre; the necrotic changes which take place convert the thrombus into a material which microscopically resembles pus. It was this septic disintegration of a thrombus which formerly induced pathologists to assume the direct entrance into and the presence of pus within the vessels. Virchow showed many years ago that the product of a broken-down thrombus is not pus, but that it is a granular *débris*, the

result of retrograde degeneration of the histological elements of the thrombus itself. When the thrombus is produced by a peripheral microbic cause, disintegration often begins between the vessel-wall and the thrombus, and the necrobiosis attending this process gives rise to early transportation of minute particles of the thrombus to distant parts of the organism, causing capillary embolism in the lungs and other organs. When a large fragment of a projecting or disintegrated thrombus becomes detached and is conveyed along the venous current to the lungs, rapid and sudden obliteration by embolism of the pulmonary artery takes place, and sudden death by asphyxia is produced, a frequent form of fatal termination in pyæmia. The thrombus, by additional deposits of the products of coagulation-necrosis, has an inherent tendency to grow in a central direction. By complete arrest of the circulation at the point of obstruction, with a diminished heart's action, the venous current on the proximal side of the clot is diminished in force, a condition which favors coagulation-necrosis, and consequently also the growth of the thrombus. A thrombus starting in a small vein in the immediate vicinity of an infected wound, by its growth in a central direction, gradually invades larger vessels, and when such is the case the projecting portion into the free lumen of an adjacent larger vein may become detached by the blood-current, and after passing through the right side of the heart is arrested in the pulmonary artery, where it causes a sudden ischæmia of the lungs and death from asphyxia. The formation of infarcts in the pulmonary artery is thus easily explained in pyæmia, but it is more difficult to account for the presence of emboli in more distant organs.

The question presents itself, How are we to explain the formation of metastatic deposits beyond the first filter, the lungs, in more distant parts of the body, as the liver, kidneys, pancreas, brain, and joints? It is impossible for the fragment of a thrombus of any considerable size to pass directly the pulmonary filter, as it is mechanically arrested in some of the branches of the pulmonary artery. The embolus under these circumstances becomes the nucleus for a new or secondary thrombus, which by extension through the pulmonary capillaries may reach directly the systemic circulation, where it serves as a new focus for the dissemination of the specific germs and furnishes the material for systemic embolism. The germs in this secondary thrombus have a more ready access into the general circulation, and by implantation upon the walls of the capillary vessels initiate a pathological condition the same as I have described in the vessels at the primary infection-atrium. I have already informed you that these so-called metastatic abscesses are not true abscesses *ab initio;* their close resemblance to pus led Virchow to apply the term "puriform" transformation in contradistinction to "purulent." He knew that while pus within the vessel was usually absent, if life was sufficiently prolonged the inherent tendency of these deposits was to produce pus, the result, as he thought, of inflammation not within the vessels, but in the tissues adjoining the thrombosed vessel. This, we have seen, is accomplished either by the transportation of infected fragments of a thrombus and embolism, or by direct implantation of the infective germs upon the capillary walls. These germs, again, reach the paravascular spaces either directly or through the agency of wandering cells, and establish here a suppurative inflammation, with the formation of an abscess around the obstructed vessel: a purulent periphlebitis or periarteritis. This is usually the pathological condition we expect to meet with in cases of slow chronic pyæmia as compared with the rapidly fatal form where there is not sufficient time for secondary metastatic suppuration to take place.

In recapitulating the etiology and pathological anatomy of pyæmia, I wish to recall once more the local formation of an infected thrombus at the site of injury or operation, the infection always being due to the presence of pus-microbes, which by implantation upon the intima produce necrosis of the endothelial cells and gradual narrowing of the lumen of the vessel by conglutination of the third corpuscle, which form a mural thrombus, complete obstruction of the vessel being finally accomplished by coagulation-necrosis. An infected thrombus, as we meet with it in pyæmia, is seldom, if ever, removed by absorption; its tendency is to disintegrate and to disseminate the first local pathological conditions by embolism.

Modern research has come to our assistance in correctly interpreting the formation of pus in parts distant from the

primary focus of suppuration. A secondary abscess can only occur after pus-microbes enter the circulation and have found favorable conditions for localization and reproduction in some distant part.

In regard to the prognosis of pyæmia, it may be safely stated that it constitutes one of the gravest complications in surgical patients. When the disease is once fully established, with very few exceptions indeed, it always proves fatal. For reasons we do not as yet fully comprehend, its duration varies greatly in different cases. It may prove fatal in a few days, or the disease may be prolonged for months; and in one of Billroth's cases the patient lived for more than a year. In the acute, rapidly-fatal form, death is produced by ptomaine intoxication from rapid, general dissemination of microbes, —a septico-pyæmia by embolism of the pulmonary artery or implication of some vital organ in the process. Fortunately, even in this dreadful disease we observe efforts on the part of the organism towards localization, and, as the few cases of recovery would tend to prove, a time finally comes when the system becomes tolerant of the germs or the germs ultimately lose their potency. It is a strange fact about pyæmia that while the clinical tendency of the disease, from the very nature of its essential cause, is to progress, the process of this gradual invasion is always attended by pathological conditions which are calculated to retard or arrest infection. In this regard we observe a marked difference between sepsis and pyæmia. In septicæmia the germs are rapidly diffused throughout the entire circulation, and we observe a complete absence of any intravascular conditions calculated to prevent this rapid dissemination. The general condition of a septic patient indicates the existence of an uninterrupted, continuous production and diffusion of ptomaines which soon results in death by toxæmia. In pyæmia, step by step, the system seems to fight against the diffusion of the microbic cause by establishing mechanical barriers in veins, capillaries, and arteries by thrombosis and embolism. This difference must be due to the intrinsic properties of the germs themselves. The microbes of pus produce local conditions favorable to the retardation or arrest of infection at the primary infection-atrium and the distant points of localization. The inflammatory changes within and without the vessel-wall produced by the presence of pus-microbes are well calculated to limit the infective process and explain satisfactorily the chronicity of pyæmia as compared with septicæmia. The pathological feature of pyæmia which determines the unfavorable result principally consists in the inherent tendency of the germs, wherever they may be, to reproduce the identical processes in various parts, and thus indefinitely multiply in the organism the primary local conditions which finally cause death from exhaustion or intoxication, while at the same time the patient is constantly in danger of death by pulmonary embolism or visceral disease of important organs. For instance, a small suppurating pulmonary infarct may produce an empyema which may become the direct cause of death. Again, a small infarct in the liver may become the nucleus of an enormous abscess of the liver, which in itself would constitute a sufficient cause of death. Finally, the transportation of a minute fragment of an infected thrombus into the vessels of the brain may lead to a cerebral abscess with a rapidly-fatal termination solely from this cause.

The danger to life from multiple suppurative polyarthritis, a frequent condition in pyæmic patients, is well known. In the absence of serious visceral lesions, it must be looked upon as a frequent and direct cause of death. While we are more familiar with the manner in which metastatic abscesses are produced in pyæmia, the occurrence of suppurative arthritis is more difficult to understand. It is possible that the synovial membrane is affected in the same manner as the peritoneum in the animals inoculated by Koch, where we have reason to believe the germs reached the peritoneum through the connective-tissue spaces. If this explanation should prove to be correct, it is evident that the occurrence of suppurative arthritis in pyæmia would indicate a grave form of the disease, as it would imply extensive diffusion of microbes beyond the boundary of vessels, and at least in many instances necessarily accompanied by multiple dépôts of suppuration in other parts of the body. As a rule, it can be said that time is an important element in prognosis. In acute cases the processes characteristic of pyæmia progress rapidly from the seat of infection

to distant parts and organs, and the disease is progressive in every sense implied by that term.

Wherever antiseptic surgery is practised thoroughly and intelligently, pyæmia seldom, if ever, occurs. As the disease can only occur after suppuration has been established, it is evident that all measures calculated to prevent suppuration also prevent this disease, and where suppuration has occurred, rigid antiseptic treatment diminishes the danger of pyæmia.

If the curative treatment of the disease has been and always will be unsatisfactory, we are now—thanks to antiseptic surgery—enabled to prevent it almost with certainty in all cases of intentional wounds. It is easier to prevent this disease than to cure it. If antiseptic surgery furnishes almost absolute protection against suppurative inflammation, you can readily conceive that it furnishes the only absolute protection against pyæmia. You must become fully aware of the importance of this assertion in the treatment of small and apparently insignificant wounds, as any suppurating wound may become the starting-point of pyæmia. The prophylactic treatment consists, in brief, in preventing the entrance of pus-microbes into wounds, and in case of contaminated wounds to effect their removal or to destroy their power of reproduction by reliable antiseptic agents, and to guard against accumulation of pus by securing free drainage. Clinical experience and scientific research corroborate the statement that pyæmia is prevented with certainty by preventing suppuration.

It becomes, therefore, your duty to perform every operation, no matter how insignificant, under strict antiseptic precautions, in order to protect the patient against the remotest possibility of incurring this disease. You must treat all accidental wounds as infected wounds, and resort to such a course of disinfection as will transform them into aseptic wounds. I have already informed you that pyæmia is very prone to follow suppurative inflammation in certain tissues and localities where the entrance of germs into veins is favored by anatomical peculiarities. The older surgeons feared wounds of veins and the medullary tissue of bone, on account of the well-known fact that when these tissues were the seat of suppuration pyæmia was liable to follow. Acute infective osteomyelitis and suppurative inflammation of the internal ear and the mastoid process are prone to be followed by pyæmia for the same reason. You will readily appreciate the importance of adopting timely prophylactic measures in the two latter conditions by exposing the seat of suppuration by operative treatment for the purpose of effecting a mechanical removal of infected tissue, and thorough disinfection with a view of preventing the occurrence of an infective thrombo-phlebitis or sinus-phlebitis and pyæmia.

In the consideration of the curative treatment the question naturally presents itself, Can anything be done after we are able to ascertain that the disease exists? Theoretically, it would appear that as long as the infected area remains sufficiently circumscribed and amenable to direct operative removal we should not despair, but should resort to prompt and efficient means to remove the infected tissues, and especially the thrombosed veins, by operative measures. Practically, it would be exceedingly difficult to ascertain whether, in any special case, the infection had not passed beyond all possible reach, and thus rendered any operation useless, and on this account unjustifiable. Where the disease takes a starting-point in a limb, as the medulla of a bone, it is only proper to remove, as thoroughly as possible, the infected tissues by scraping and ignipuncture, or by resorting to a timely amputation. In suppurating wounds not admitting of such heroic measures, the surgeon must content himself with securing free drainage, or with resorting to permanent antiseptic irrigation with a one- or two-per-cent. solution of acetate of aluminum. It is only proper to insist that the parts the seat of thrombosis should be left perfectly at rest, so as to prevent the detachment of fragments by mechanical violence, an accident which is always followed by grave consequences. In all cases where internal metastatic deposits have taken place, it is too late to remove, with any hope of success by operative treatment, the infected tissues at or near the primary place of infection, as the disease will in all probability multiply itself from these distant centres. In such cases it is advisable to support the heart's action with large doses of alcoholic stimulants, from one to two pints of brandy daily, with a faint hope that, by prolonging life for a sufficient

length of time, the disease may, as it were, exhaust itself, and the patient recover after a long and lingering illness. In all pyæmic patients it is advisable to give a daily antipyretic dose of quinine during the remissions, and to support the strength of the patient by concentrated, nourishing liquid food.

ORIGINAL COMMUNICATIONS.

CLINICAL VALUE OF ANTIPYRETICS.*

BY LEROY J. BROOKS, M.D.,
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IT has been demonstrated that physiological heat-production is the result of metabolic changes in the tissues, and that a delicate balance is maintained between production and dissipation by heat-regulating centres.

Wood finds a heat-producing centre near the conical sulcus of a dog, while Aronson and Sachs locate it near the inner side of the corpus striatum. The pathological evidence consists in the fact that crushing the cervical medulla produces a most exorbitant rise of temperature. Cases are on record of a temperature of 122° produced by injuries to the spinal cord. Moreover, a more intimate study shows that total section of the cord near the pons produces a rise of temperature, but that if the anterior columns are spared such a rise does not take place.

In divisions of certain portions of the cord which especially influence the vasomotor system, as well as in disease of the same portions, there is produced a marked lowering of temperature. The question is of importance to us in determining whether pathological heat-variations are the result of a disturbance of the physiological heat-regulating powers through impressions upon the nerve-centres, or some foreign element introduced into the blood has elevated the temperature by an increase of the tissue-changes. In these days of microbes we find that the claim has been made that fever-heat is the result of a struggle between the system and certain microbic foes.

The suggestion leads at once to the inference that if temperature-elevation be due to changes produced by the presence of bacteria in the blood, or if the fever is the result of some poisonous secretion or material produced by bacteria or of some specific poison acting directly upon the blood, then such remedies as would destroy the activity or life of bacteria, or of such specific poisons, would reduce the temperature. Were the theory a correct one, then antisepsis and antipyresis would be synergistic and synonymous terms.

It leads us to ask, Are antiseptics, then, antipyretics? If so, are they antipyretic because of their antiseptic qualities or because of some other specific action of the drugs?

As we investigate, we find that many of the strongest antiseptics are the feeblest antipyretics.

We need but to mention corrosive sublimate, which is fatal to lower organisms in one part to twenty thousand,—which is ten times more powerful than thymol and benzoate of soda, twenty times as strong as creasote, thyme, and benzoic acid, thirty times as powerful as salicylic acid and eucalyptol, and one hundred times as powerful as carbolic acid and quinine as an antiseptic,—yet no specific antipyretic effect can be attributed to it. 2d. Some of the most active antipyretics are feebly antiseptic,—instancing quinine, carbolic acid, and the benzoates. 3d. As a rule, it is impossible to introduce into the blood of a living organism, and maintain the presence for any considerable time, of a sufficient amount of an antiseptic drug to destroy the activity or life of bacteria therein. It is probable that corrosive sublimate is the one agent that could attain nearer this end than others, but this it could not accomplish without extreme toxic effect.

Again, it is maintained by many that bacteria may exist in healthy, circulating blood (doubtful); that they may exist in diseased conditions without causing an elevation of temperature, as in diphtheria, afebrile pneumonia, hydrophobia, and malarial diseases; and that, while the actual abundance of bacteria in the blood may bear a relationship to the severity of the disease, it does not to the degree of temperature-elevation.

The antipyretic qualities of antiseptic drugs are not manifest until a secondary effect is produced,—namely, a profound impression upon the central organs.

* Read before the New York Medical Association, Third District Branch, at its annual meeting held June 16, at Elmira, New York.

To give the specific authorities for these statements would lead us too far from the purpose of our paper. We then venture the statement that antisepsis does not necessarily mean antipyresis, and present the following class of antipyretics: first, those that retard heat-production; second, those that diminish heat-production and increase heat-dissipation; third, those that increase heat-dissipation. The first class includes quinine; the second, the salicylates, antipyrine, antifebrine, thalline, kairine, hydrocinchonine, and others of the same derivation; the third, aconite, gelsemium, veratrum, and similar remedies, to which may be added cold applications.

Quinine is the most prominent, and it may be said to be the only drug that by its direct specific action diminishes the production of heat alone. This cannot be due to its effect upon malarial poisons, for we find that it will reduce temperature with equal force in pneumonia, typhoid, erysipelas, eruptive diseases, surgical fevers, etc., which do not possess any malarial element.

It cannot be due to its antiseptic qualities, for Binz and others have shown the minimum quantity of quinine necessary to prevent the development of bacteria to be one part to one thousand. It is evident then that with an average of eighteen pounds of blood, it would require one hundred and thirty-eight grains of quinine circulating therein to meet such conditions. Quinine is discharged at so rapid a rate from the blood that we may readily conclude that it would be impossible to administer with safety to the patient a sufficient quantity to affect the bacteria.

Quinine lessens tissue-metamorphosis and diminishes the activity of white blood-corpuscles, but to that cannot be ascribed its profound antipyretic effect. It will lower temperature in the healthy body. It will reduce temperature without a marked exhibition of effect upon the arterial or respiratory system, and without exciting perspiration. We conclude, then, the effect of quinine upon the temperature-elevation is the direct result of a direct and specific action upon the heat-producing centres. We are confirmed in this when we find that it is the only drug which will completely and permanently act upon heat-production so as to prevent the recurrence of fever non-malarial in its origin.

The specific effect of the single dose of quinine on temperature begins in two to three hours, reaches its maximum in five to six hours, and continues from twenty-four to seventy-two hours, and the reduction is sometimes permanent.

The second class, those which both diminish production and increase dissipation,—of which the salicylates, antifebrine, and antipyrine, and drugs of the same derivation may be considered as types,—possess the following peculiarities in general: 1st, their effect upon heat-production is less profound and more variable than quinine; 2d, they rarely destroy the tendency to overheat-production; 3d, they increase heat-dissipation; 4th, except in extreme doses or previous cardiac weakness, they show but a moderate effect upon the heart or respiratory organs; 5th, except where idiosyncrasies exist, their specific effect upon the body-temperature is the result only of sufficient dosage to produce a profound or toxic impression upon the nerve-centres.

The salicylates are second only to quinine, possessing a greater power over temperature-production than antipyrine and its class, and more influence over heat-dissipation than quinine. They rarely destroy the tendency to recurrence of fever, though more often than the others of the second class. They have a moderate effect only upon the heart and arterial tension or respiratory organs. While salicylic acid is strongly antiseptic, its combinations with alkalies are not.

It has been proved that salicylic acid unites with the soda of the blood to form salicylate of sodium. (Bartholow.)

We may reason, therefore, that their antipyretic qualities are not due to their antiseptic powers. Salicylate of sodium will often produce a fall of temperature without causing sweating. (Ross and Nothnagel, p. 468.)

There is rarely any serious after-effect from its administration, except after giving extreme doses. (*Idem*, p. 475.) To obtain antipyretic results large and frequently-repeated doses should be given, when the effect upon temperature begins in one to two hours, reaches its maximum in three to six hours, and generally ceases in twenty to twenty-four hours.

Antipyrine and antifebrine may be taken as types of the remainder of this class. In dosage antifebrine is about twice the strength of antipyrine. Both are easy of

administration. Their specific action upon temperature is temporary, commencing in one-half to two hours after administration of proper dose, reaching the maximum in three to five hours, the effect being then rapidly dissipated. They probably do not prevent the recurrence of a specific fever; they do not possess antiseptic qualities sufficient to influence the condition of the blood, and no perceptible effect upon the white blood-corpuscles; their effect upon heat-dissipation is much more pronounced than with the salicylates; they have a positive effect upon the nervous system, allaying headache, producing sleep, and quieting delirium; they influence the vaso-motor system; they increase perspiration and urine. Serious after-effects, except with extreme doses or previous cardiac weakness, have not been noted. They diminish arterial tension moderately. An occasional measle-like eruption has been noticed as being produced by antipyrine, which readily disappears when it is discontinued. The drugs are applicable to any disease with temperature-elevation.

The third class includes, as we have already stated, aconite, gelsemium, veratrum, and cold. They have no specific action on temperature-production, and centre their effects upon heat-dissipation. They tend to paralyze the heart and respiratory organs, reduce arterial tension, and are sedative to the vaso-motor system. It is claimed that they produce first an increase in the actual temperature by the influx of blood in the paralyzed vascular fields, then a sluggishness of the current, greater evaporation, and a resulting cooling of the surface. A hot bath, antimony, an emetic or cathartic, may reduce temperature in the same manner as other motor-paralyzers; cold acts in a more mechanical way by abstraction of heat. The effects of the class upon increased temperature are temporary, disappearing as soon as the drug-effect has ceased. They are applicable to sthenic cases,—acute febriculæ, inflammations,—and act where doubtless heat-production is normal and heat-dissipation diminishes.

Practically, then, we arrive at the following conclusions concerning pyrexia. Fever-heat is a loss of balance between the producing and dissipating heat-functions. There may be an increased production with dissipation normal, or diminished dissipation with production normal, or other combination of the different conditions. Thus we may have an actual oral temperature of 104° without increased production, which in itself would not be serious; on the other hand, increased production may concur with increased dissipation, not indicating a high temperature-elevation, yet being disastrous in its effects upon the patient. We not infrequently see this in adynamic diseases, where there occurs the prostration, subsultus, delirium, rapid emaciation that we would naturally associate with intense fever, yet without marked increase. Again, we see the further extreme where, production remaining normal or subnormal, an intense impression upon the nerve-centres—the vaso-motor paralysis, with cardiac and respiratory depressions,—we get the excessive dissipation and the resulting collapse of cholera, the nervous shock, the cold stage of ague, the congestive chills, the collapse of yellow fever, etc.

McAllister, in his recent studies of fevers, demonstrates that in fevers there is disturbed, first, the thermotaxic, or heat-regulating; second, the thermogenetic, or heat-producing; and, lastly, the thermolytic, or heat-losing functions. In the beginning of a fever, then, the important indication is to restore, if possible, the disturbed thermotaxic and thermogenetic functions: to give a drug, if we possess one, that will prevent, modify, or shorten the disease. It is not necessary to enter literature, nor illustrate by cases, except to say that an analysis of the opinions of Liebermeister, Niemeyer, Reynolds, Da Costa, Flint, Cleveland, and others, compels the conclusion that quinine aborts some, shortens the duration of others, and ameliorates most cases of fever.

In the advance of the fever it presents a somewhat different aspect. With the hope of aborting the disease gone, the thermogenetic and thermolytic functions disturbed, more or less tax upon heart and respiratory organs and the nervous system involved, we look for remedies where production of heat can be diminished without serious depressions, the elimination increased, and the nervous system quieted, and we find that the salicylates, antipyrine, and antifebrine meet most admirably these needs. Every-day experience proves this beyond peradventure if, as we have no reason to doubt, "the dangers of fevers are more from the prolonged and continuous temperature-

elevation and the resulting disaster to the bodily tissues" than from any other cause. The speedy dissipation of the antipyretic effect is preferable to the more enduring and profound, but proportionately depressing—at this stage—effect of quinine. Again, quinine in antipyretic doses increases cerebral disturbances. Antipyrine and antifebrine allay delirium and possess a distinct hypnotic action. Out of eighteen cases of typhoid fever in which antipyrine or antifebrine was used, we recorded two instances of apparently undue depressant action as a direct result of the drugs. In each instance the administration was towards the close of the fever,—one patient 9 years old, the other 18 years. In the younger the temperature had been normal for three days; through an excitement it rose to 103.5°, and the mother gave ten grains of antipyrine. I found the patient with feeble pulse, some cyanosis, cold extremities, nausea, temperature 97°. A few doses of carbonate of ammonia and whiskey brought a quick restoration. In the second the temperature had risen to 103°, when ten grains of antipyrine were given by the nurse, and the dose repeated in an hour without taking the temperature. The symptoms an hour later were similar to those of the first case, and, like that, soon responded to stimulants.

The lesson of the two cases is that, at the closing stages of a fever, the order is reversed, and we have, first, a restoration of the thermolytic; second, the thermogenetic; and, lastly, the thermotaxic power. Just here nature tries to conserve her forces for the natural reaction of fevers, when the taxed muscles, the weakened heart and respiratory organs need the most judicious management. To give at this stage an antipyretic—that is, to diminish production or create an over-elimination—might be easily disastrous. We should withdraw all antipyretics, and resort to stimulants and tonics. Cold should be used in the earlier and middle stages of a fever, where by its mechanical effect it assists in the general purpose of fever-reduction and subserves the comfort of a patient.

From the records of the eighteen cases referred to in which antipyrine was used, we note that there were two cases of intestinal hemorrhage: one several days after convalescence had begun, and nearly ten days subsequent to any use of antipyrine, caused the patient's death. In the other hemorrhage was slight and of short duration, and had no relation to drugs administered. There were two deaths: one from hemorrhage, as above stated, and one from excessive cerebral complications and previous bad condition.

Occasionally a peculiar susceptibility exists, and full effect is obtained from small doses; and, again, in some instances the maximum doses were required to get the desired result. We found but one case where the measle-like eruption appeared.

The third class of antipyretics should only be used in the active sthenic stages.

The typical treatment of fevers should be:

First thermometric stage: abort; modify; shorten duration by quinine assisted by heat-dissipaters.

Second thermometric stage: keep temperature below 103° by salicylates, antipyrine, antifebrine, assisted by cold applications, lowered surrounding temperature, hydrogenized food.

Third thermometric stage: feed; stimulate; use nerve-sedatives and tonics.

Lastly. The antipyretic effect of quinine commences about the third or fourth hour; reaches its maximum in the sixth or eighth hour; continues its effect from twelve to seventy-two hours, and sometimes permanently. Salicylates commence the second or third hour, reaching the maximum in four to six; and continue their effect twelve to fifteen hours. Antipyrine, antifebrine, and this class commence their effect in one-half to one hour; reach the maximum in two to three hours; continue their effect three to six hours. Aconite, gelsemium, etc., commence their effect within the first hour, reaching the maximum in from two to five hours; the effect dissipates soon after ceasing administration of drug.

NEW METHOD OF MANAGEMENT OF THE PATELLA IN AMPUTATIONS AT THE KNEE-JOINT.

BY Z. H. EVANS, M.D.,
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ON the 13th day of August, 1872, I was called to see Rev. Harvey Peirce, aged 65, minister of the gospel, who had just sustained a compound comminuted fracture of the upper third of the leg, implicating the knee-joint, caused by the kick of a horse. The case being one demanding

amputation at the knee-joint, I proceeded at once, with the assistance of Dr. Hosmer, to operate by making long posterior and short anterior flaps (Hoin's method).

In making my flaps I cut the posterior too short, so much so that if I had removed the patella I would still have lacked sufficient tissue in my anterior flap to have brought it in proper apposition with the posterior. In this extremity I decided to excise the condyles at their face, which having removed, my flaps came nicely together, bringing the *patella immediately over* the end of the shaft. The patient made a speedy recovery, riding out to church in a few weeks from day of operation. In a subsequent case of exsection of the knee-joint followed by gangrene of the foot, making it necessary to remove the leg, I operated as in my first case (the condyles having been removed in the first operation by exsection), with good result. The patient died a year after with apoplexy, and upon post-mortem examination of the stump I found the patella firmly united to the end of the femur by *bony* deposit. I am of the opinion that bony union of the patella and end of the femur will take place without sawing through the lower surface of the patella, as practised by Gritti and modified by Stokes and others. I am disposed to believe that, by reason of the greater firmness and less liability to tenderness of the stump, also in the degree of retention of the purchase of the quadriceps muscle, this operation is preferable to those recommended in our late works on surgery. By this method the leg retains its full strength for future use in the adaptation of an artificial member.

THE USE OF COLD WATER IN THE TREATMENT OF TYPHOID FEVER, WITH TWO CASES.

BY S. P. ALLEN, M.D.,*

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WITHIN the last few years much has been said and written, I am aware, upon the use of so-called antipyretics in the treatment of typhoid fever, and, if properly used and carefully watched, they are undoubtedly of great importance in the treatment of these cases. But, on the other hand, if used indiscriminately and by way of routine, they may be, and no doubt have been, worse than no treatment. I think that it is admitted by all at the present time that typhoid fever belongs among the so-called self-limited diseases, and that the majority of cases occurring in civil practice where the temperature does not rise above 103° would recover without much if any medication further than absolute rest, with plenty of good air and careful diet.

That there are exceptions to this rule I am free to admit, and it is in just those cases that the antipyretic treatment is capable of being useful and doing most good.

There have been various remedies used for this purpose, and no doubt all have done some good, and each have their advocates. Among those that have been used most I might mention quinine, antipyrine, and cold water. But it is only of the latter that I wish to speak, and I will give the history of two cases among the many that have occurred in my own practice, and that were treated mainly by the use of cold water. I select these cases because of the extreme high temperature that prevailed during so much of the time in the course of the disease.

Case I.—October 11, A.M. Was called to visit A. P., aged 21, by occupation a farmer, and all his life up to this time had been strong and healthy and accustomed to hard work. He had been sick five days when I first saw him, and at this time the following symptoms were present. Epistaxis had been quite persistent, at times, for three days; face dark, almost purple; slight cough; iliac tenderness; ochre stools; drowsy, with more or less muttering when not disturbed; severe headache, also complained that the light hurt his eyes; tongue dry, with heavy, dark coat; pulse 100, temperature 103°. Treatment: ordered him to be sponged with tepid water every two hours. I gave as a sedative aconite and spirits of nitre.

October 12, 9 A.M. Temperature 104°; other symptoms about the same. I then wet a sheet in water at 70°, folded it and wrapped it around his body, and used water at 65° to sponge his limbs and arms, and at intervals of fifteen or twenty minutes would sprinkle water at 65° on the sheet. Continued this for two hours, when temperature was 103°; left sheet as it was, and after one hour temperature was 102°. I left the thermometer with nurse, and, after instructing her how to use it, told her to use the water every two hours if the temperature should exceed 103°, and by this

* Read before the New York State Medical Association, Third District Branch, at the annual meeting held June 16 at Elmira, New York.

means we succeeded in keeping the temperature at 103° or less for eight days.

October 19, 8 A.M. Called and found that he had passed a very restless night; had had considerable hemorrhage from the bowels, and had been very delirious; tympanites had increased considerably; pulse 115, temperature 106°; and because of these symptoms the nurse had neglected to use the water since midnight. After looking the case over carefully, I resolved to persist in the use of the cold water, thinking that if I could again reduce the temperature I might yet possibly save my patient. Accordingly I concluded to wrap him in the sheet as before, with water at 65°, and left but little else over him, and told the nurse to sponge his arms and limbs, and every fifteen minutes to wet the sheet with cold water. This we continued for four hours, when temperature was 104°. I then removed the sheet, wiped him dry, and left him for four hours. 4 P.M., temperature 104°; no more hemorrhage from the bowels; pulse 105; sleeping; had used turpentine and opium for bowels. 8 P.M., temperature 104°; used cold water same as before for one hour, when temperature was 103°; left sheet around him, and ordered arms and limbs to be sponged every two hours during the night; gave two grains of quinine every four hours, with tablespoonful of whiskey in glass of milk between.

October 20, 9 A.M. Pulse 95, temperature 102¼°; removed sheet, which was dry now, and ordered sponging to be continued every two hours; other treatment same as before. 6 P.M., temperature 103°; continued same as before.

October 21, 9 A.M. Temperature 102°; no more trouble with bowels; less delirium; pulse 90; tongue beginning to moisten; continued as before, except opium and turpentine. 5 P.M., temperature 102½°; bowels moved twice, no blood; pulse 88; continued as before.

October 22, 11 A.M. Temperature 101½°; one movement of bowels during night; pulse 85; continued quinine and whiskey, and sponging every four hours unless he asked for it oftener, for he had begun to want it: said it made him feel better. 6 P.M., temperature 102°; continued same as before.

October 23, 9 A.M. Temperature 100°, and it did not again exceed that.

I continued the quinine and whiskey for another week, with plenty of milk and beef-tea, at which time I considered the case comparatively out of danger and discontinued my visits. The case went on favorably and made a good recovery.

Case II.—April 6, was called to see J. B., aged 18 years. Had been sick then three weeks, under the care of a neighboring physician.

I found the following symptoms present:

There had been more or less hemorrhage for several days, but for the last few hours it had been more profuse, and with an evacuation which occurred just as I came to the house I should think there was at least half a pint of fresh blood. Tympanites was well marked; pulse 130; temperature 106¼°, with low, muttering delirium when not disturbed; tongue moist, but very red.

I suggested cold water externally, with the use of opium, whiskey, and turpentine internally.

The family, thinking that the patient must die, were willing to try anything; accordingly, a wash-tub half full of ice and water was brought in the room, and, taking two sheets, I folded them so that they would cover the body from the pubes to the breasts. After laying one on the ice until it was cold, I proceeded to cover the whole anterior of the body, and placed the other on the ice. They were changed every fifteen minutes for an hour, and after that every thirty minutes for two hours, when the temperature was 103°. I then ordered them to be changed every hour, and left for four hours. On my return I found temperature 101½°; no hemorrhage; pulse 118; patient sleeping quietly.

I then ordered them to use the water at about the temperature of the room, and change every two hours. Continued opium and whiskey, and also two grains of quinine every four hours.

This was followed closely for one week, except twice for a few hours, when we again used the ice-water, in order to hold the temperature at or about 103°.

At the end of the week temperature was 100°, and did not exceed that again.

Convalescence in this case was slow, yet the patient made a good recovery.

Since that time I have treated several cases in the same way, without losing a single uncomplicated case, and I have seen convalescence well established as early as the eighteenth day. I feel assured that in the majority of these cases, if we can hold the temperature below 102½° or 103°, very little other medicine will be required.

The following conclusions which were given by Dr. Austin Flint, after closely watching the effects of cold water externally in the treatment of seventeen cases of typhoid fever that came under his care in Bellevue Hospital some years ago, are sustained by the results in thirteen cases that occurred in my practice since that time:

1. That by the use of cold water externally in cases of typhoid fever the temperature of the body may, after a variable time of its continuance, be reduced to 102°, or even lower.

2. After a period varying very much in different cases, and also at different times in the same case, the temperature rises as high or higher than before the reduction.

3. Upon repeating the employment of cold as often as the axillary temperature exceeds 103°, the number of repetitions necessary is extremely variable in different cases.

4. The sponge-bath, with the wet sheet and sprinkling, may be employed to the exclusion of the bath-tub in the treatment of typhoid fever.

5. These modes of employing cold water may be continued sufficiently long for the reduction of the temperature to 102°, or even lower, and repeated as often as may be required, without fear of injury. And the study of these cases furnishes no ground for supposing that a liability to complications or accidents is thereby increased; and that the reduction of the temperature by these modes, as often as it rises above 103°, improves the condition of the patient.

6. The results of the analysis of those cases where cold has been faithfully used afford us encouragement to employ it with the expectation of diminishing the severity of the disease and its danger to life.

TRANSLATIONS.

THE CONSTIPATION OF PYREXIA.—Experimental researches of Professor A. Bokai refer the constipation of the bowels which usually accompanies fevers to the effects of the elevated bodily temperature upon the nervous system.

The conclusions drawn from the series of observations are as follows (*Archiv für Experimentelle Pathologie und Pharmakologie*, August, 1887):

1. The reduced amount of nourishment taken by persons sick with fever may favor the occurrence of constipation, but the condition is especially explained by the fact that during the course of the fever the inhibitory nerves of the bowel are in a condition of increased irritability.

2. The exalted irritability of the inhibitory nerves of the bowel during the fever is connected with the increase of the bodily heat, and is a result of it.

3. If the temperature in the rectum, in animals, be raised (by Bernard's apparatus) to 39° C., and not over 42.5° C., the irritability of the inhibitory nerves of the bowel is always evident, and can be reduced by small hypodermic injections of morphine.

4. A bodily temperature going higher than 42.5° C. brings about a paralysis of the inhibitory nerve-fibres of the intestine.

TREATMENT OF SYPHILIS BY HYPODERMIC INJECTIONS OF PHENATE OF SODIUM.—Dr. K. Schadek (*Medicinscoie Obosrenié*, No. 6) reports a new treatment for syphilis. Last year Professor Gamberini proposed to treat syphilitic infection by the double antiseptic, the phenate of mercury. This is a yellowish powder, having a very decided carbolic acid odor, which is obtained by mixing a solution of corrosive sublimate with one of phenate of potassium. This was given by Gamberini in pills of a thirtieth of a grain, of which from two to six were given each day. Drs. Schadek and Troitzki have also used it by injections both subcutaneous and deep.

The injections were not painful, or only slightly so. A two-per-cent. solution in an emulsion of gum arabic was ordinarily used for subcutaneous injections. Dr. Troitzky found that ten injections were sufficient in the majority of cases to cause the syphilitic phenomena to disappear.

In résumé, the author found that the phenate of mercury is superior to all the other mercurial preparations employed by injections. This superiority is due to (1) the rapidity of its action when administered in this form, (2) the absence of any pain or tenderness in the tissues after the injections, and (3) the relative rarity of unfortunate accidents such as stomatitis growing out of the use of the mercurials.—*Bulletin Général de Thérapeutique*, July 30, 1887.

LIGATURE OF THE THYROID ARTERY IN GOITRE.—At the recent German Congress of Surgeons, Dr. Woelfler reported the case of a woman, æt. 45, in whom this operation was done in consequence of an error of diagnosis. The patient came to the clinic with a high grade of dyspnœa, and said that the goitre which was present had been of the same size for years; later it proved itself to be carcinoma. The recovery was without a bad symptom, and the patient left the institution in four weeks. The dyspnœa was gone, and the circumference of the neck was less. In four weeks, however, the difficult breathing returned and the patient came again to the hospital, where a little later she died. On post-mortem examination the author found that the right half of the thyroid gland, in consequence of a necrobiotic process, was notably diminished in size.

PHILADELPHIA
MEDICAL TIMES.

PHILADELPHIA, SEPTEMBER 3, 1887.

EDITORIAL.

THE NINTH INTERNATIONAL MEDICAL CONGRESS.

FROM the time the announcement was first made to the world that the International Medical Congress at Copenhagen had formally accepted the cordial invitation extended by the American Medical Association on behalf of the profession of this country to hold its next meeting at Washington, this journal has never lost faith in the success of that meeting, nor failed to advocate it. As might have been anticipated, it has given rise to some heart-burnings in the profession here, particularly among a certain clique who early tried to monopolize all the offices, but happily the attempt was frustrated; the American Medical Association proved equal to the great emergency, and a proper preliminary organization was effected. By this time the few disaffected ones, who exerted themselves to the utmost in order either to prevent the Congress from meeting in this country at all or to make it a failure by inducing all the prominent European members to remain at home, have probably seen the error of their ways, and are applicants for admission to the Congress which they sought to destroy. Let the cloak of charity be thrown over them, and let them come in: the demonstration of the trifling amount of influence which they were able to exert may afford them a useful object-lesson.

The medical journals, however, which opposed the Congress are in a different position. They abused the trust reposed in them, and deliberately did all in their power to discourage and defeat the efforts of those who were laboring to establish the foundation for a successful meeting. It cannot be expected that they will be allowed to get off so easily. Their offence cannot be readily forgotten nor forgiven. Their publishers can best tell the results of incurring the censure of the profession.

But these questions are not for our guests. We bid them cordial greeting. They shall be met with welcomes wherever they choose to go. Entertainments in New York and Philadelphia have already been given to members on their way to the Congress. During the coming week engagements are announced on the programme as follows: Monday evening, from 8 to 11, conversazione in the large United States Pension Hall at Washington; Tuesday evening, visit to the Corcoran Art Gallery; Wednesday evening, from 8 to 12 o'clock, reception by the citizens of Washington; Thursday evening, from 8 to 11, general reception and buffet banquet at United States Pension Hall; Saturday afternoon, a visit to Mount Vernon. The whole to terminate with an excursion to Niagara Falls.

The scientific papers are numerous and varied, and the indications certainly are in favor of a Congress which shall not be inferior to its predecessors in interest or value.

A MEDICO-LEGAL DECISION AFFECTING LIFE-INSURANCE.

THE insurance companies are frequently found contesting the payment of policies of deceased members, and in a recent case the United States Mutual Accident Association refused to pay a policy where the assured died with malignant pustule. It appeared that the deceased was employed at a place where hides and cattle were received, and that the cause of death was "a putrid animal substance on the exterior of the body working inwardly, and usually communicated from the bodies of animals suffering with diseases of the hair." The policy insured

against death from "external, violent, and accidental means," and excepted death "by taking poison."

The question was whether there could be a recovery under the policy, and the court decided that there could be. The reasoning of the court is interesting, and we quote as follows, at length:

"The word 'poison' therein is used in the ordinary meaning of a substance taken internally, seriously injurious to health, and often fatal to life.

"'Death by poison' is well understood in ordinary language. It is a phrase which would never be applied to death from a rattlesnake-bite, although that injects into the circulation what may be called a poison.

"In certain cases death comes from what is called 'blood-poisoning.' One who dies from a bullet-wound may die of 'blood-poisoning,' but he does not die from poison in the ordinary sense of the word.

"The insured died from what is known as malignant pustule. This is produced invariably, according to the testimony, 'by the infliction of animal substance upon the body,'—by the accidental deposit of this putrid and poisonous animal substance.

"This is usually communicated from the bodies or skins of animals which are suffering with this peculiar kind of disease.

"The substance may be carried by flies that have been feeding on it. The disease may come by the application of the person himself, of his hand, which has been in contact with this substance, to some abraded surface of his body; and the substance may be absorbed by the thin portions of the lips, though they are not abraded.

"The means through which deceased came to his death were external. The positive testimony of the physician shows this. The putrid animal substance reached the body, not through the stomach or lungs, but through the skin, the external covering. The cause was external as much as the crushing under a car or the bite of a rattlesnake would have been."

The gist of the decision is that, in a death of the character mentioned, the deceased could not be said to have "taken poison."

LEADING ARTICLE.

THE GUIDING PRINCIPLES OF TREATMENT IN EXCESSIVE CORPULENCE.

IT must be apparent to every observant physician that cases of excessive corpulence are becoming more frequent in this country. One reason for this greater frequency is probably to be found in the increase in number of persons who live a life of ease, who have no business or occupation to stimulate mind or body to exercise, and who have abundant means to gratify their tastes and inclinations in every particular. Most cases of acquired corpulence will be found in this class after middle age, while the hereditary tendency to excessive fat-production usually manifests itself earlier in life.

The first writer who clearly formulated the causes of corpulence was Brillat-Savarin, the brilliant author of the "Physiologie du Goût." He gives the following as causes of excessive accumulation of fat in human beings: heredity, excess of starchy and saccharine foods, too much sleep, too little exercise, and intemperance in eating and drinking. Although many advances have been made in our knowledge of food-assimilation and metabolism in the last half-century, the shrewd observations of this man of the world upon the nature, causes, prevention, and treatment of corpulence have not been superseded by modern physiological research.

Fat-tissue is formed from each of the three classes of materials which constitute the food of man,—namely, fats, proteids, and carbo-hydrates.

Animal and vegetable fats contained in the food are absorbed by the intestinal mucous membrane. Through the agency of the bile, which moistens the villi and emulsifies the fat, the latter may be taken

up and deposited as fat without first undergoing decomposition in the intestinal canal. Evidence in favor of this form of accumulation of fat has been furnished by Lebedeff and Munk. These observers fed animals upon special fats (such as rape-seed oil), and afterwards demonstrated the special fat used in the feeding experiments in the fat-tissue of the animals.

It is a curious fact that fatty acids may perform the same function in nutrition as the neutral fats. A reconstitution or synthesis of fat from fat acids and glycerin is supposed to take place in the absorbent vessels, thoracic duct, or tissues. The nutritive properties of the fatty acids have even been taken advantage of for therapeutic purposes, and it has been found that equally good results have been obtained from the administration of pure fatty acids, as palmitic and oleic, or their combinations with alkalies, as from the fats,—*e.g.*, cod-liver oil or similar nutrients themselves.

A second source of fat in the body is the decomposition of proteids. It has been shown by a number of investigators that the consumption and assimilation of food containing no fat results, nevertheless, in the formation of fat-tissue and fatty secretions. Thus, the fat contained in milk is largely in excess of the fat taken in with the food. It must therefore necessarily be formed in the body out of non-fatty materials. Carnivorous animals (dogs) deprived of fatty food, and nourished almost exclusively on proteids, yet produced fat. The formation of the higher fatty acids, palmitic and oleic, from decomposing meat-powder freed from fat, has been demonstrated by Salkowski. The process of fatty degeneration of muscular tissue is strong evidence in favor of the conversion of proteids into fat. This evidence is very much strengthened by the circumstance that in acute phosporus-poisoning the rapid fatty degeneration of proteid tissues which takes place in this condition is accompanied by an enormous excretion of urea, showing great destruction of albuminoid tissue.

The production of that curious substance known as adipocere, under certain conditions of albuminoid decomposition not yet well understood, also seems to speak in favor of the derivation of fats from proteids.

Liebig gave the weight of his great authority in favor of the view that the carbo-hydrates are the most fertile, if not the only, source of fat in the body. As pointed out above, the great physiological chemist had been anticipated in this opinion by the gastronomer nearly a quarter of a century.

For many years this view of the principal derivation of fats from carbo-hydrates held sway in physiology, but the experiments of Pettenkofer and Voit seemed to disprove the direct conversion of carbo-hydrates into fats, and it came to be accepted and taught that "no fat is produced directly from the carbo-hydrates consumed."* Recent experiments have, however, shown conclusively that, although the quantity of fat derived from carbo-hydrates is limited, this source of fat-production cannot be excluded. These food-materials have indirectly a large influence in the accumulation of fat by reason of their easy decomposition, which enables them to act as conservators of other food-constituents, which latter may be converted into and deposited as fats. The carbo-hydrates may therefore be principally looked upon as economizing the consumption of proteids and fats.

While it is probable that proteid tissue can only be built up by the proteid substances contained in the food, yet when the food consists exclusively or principally of the latter, the decomposition of proteids in the body is so great that actual loss of proteid tissue results. This has been well shown by an experiment of Rubner. He consumed for several days nearly three pounds of meat per day, but found that, after the second day, the nitrogen excreted in the form of urea exceeded that taken into the body in this enormous quantity of meat. In other words, on an exclusively proteid diet, although excessive, the proteid waste was greater than the income. But the addition of a moderate quantity of fats and carbo-hydrates reduces the proteid waste, and there is consequently a proteid gain. This is an important point to be remembered in combating corpulence by dietetic means.

These facts may be briefly summarized as follows:

Fat-tissue may be derived from (*a*) fats, (*b*) proteids, and (*c*) carbo-hydrates.

Fat consumed with the food is probably the principal source of fat-tissue.

* Landois: Lehrbuch der Physiologie, 8te Aufl., 1881.

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Cholera Morbus,
Cholera Infantum,
Summer Complaint,
Sick Stomach,
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Bicarbonate Potassium.—Antacid; corrects the acidity developed in the stomach and bowels in enfeebled states of digestion; increases the alkalinity of the blood and the oxidation and excretion of effete matters.

Pancreatin.—The albuminoid principle of the Pancreas;—promotes intestinal digestion; transforms starch into sugar and dextrine; converts proteids into peptones; emulsifies fat; curdles the casein of milk; and in general exhibits all the important functions of this organ. In the presence of an alkaline solvent this action is largely increased.

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Spiritus Vini Gallici;—Simple Elixir with Oil Mentha Piper. This menstruum will especially commend itself to physicians.

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The next most prominent source of fat tissue is proteid food-material.

Carbo-hydrates are converted into fat to a limited extent, but are principally useful as oxidizing and conservating foods.

The normal balance between proteid income and expenditure cannot be maintained on proteid food alone. Fat and carbo-hydrates are required to prevent proteid waste.

Bearing these facts in mind, we may proceed to inquire under what circumstances fat is accumulated in excess in the body.

The standard diet for an average man who does a moderate amount of work has been fixed by Voit at one hundred and eighteen grammes proteids, fifty grammes fat, and five hundred grammes carbo-hydrates. Under this diet the balance between income and expenditure, both of nitrogenous and non-nitrogenous constituents of the body, should be maintained. If, however, the individual whom this just suffices to keep in equilibrium were required to perform excessive labor, or any one of the classes of foods were reduced, he would lose weight, and probably strength. If, on the other hand, he would maintain perfect rest, or either the proteids, fats, or carbo-hydrates were increased, he would, other things being equal, accumulate fat. Should the proteids consumed be diminished while the non-nitrogenous foods are increased, an increase of fat with diminution of proteid tissue would result. If excessive, the form of corpulence known as the anæmic is produced. In this form there is progressive diminution of muscular tissue and red blood-corpuscles, and increase of adipose tissue.

It is well known that anæmic individuals are very liable to become corpulent. Chlorosis and corpulence are not infrequent concomitants. Stock-raisers are familiar with the influence of anæmia upon fat-production, and take advantage of it to fatten their stock by frequently bleeding the animals. It is also often seen that persons rapidly become fat after suffering large losses of blood. In the latter case the fat-production is promoted by prolonged confinement to bed.

Too much sleep and too little exercise promote the accumulation of fat, because the excess of fat over that needed for combustion is deposited as fat-tissue. More active exercise would burn up this excess. The experiments of Fisk and Wislicenus and of Frankland have shown that excretion of carbonic acid and the production of heat during work are greater than could be accounted for by decomposition of proteid tissues. Hence it is necessary to assume that an increased destruction of non-nitrogenous constituents of the body takes place during active exercise. Pettenkofer and Voit have shown this by actual experiment, and the latter investigator has estimated that during ten hours of hard work eighty-two grammes more fat are used up in the body than during the same period of rest. The great importance of a knowledge of this fact will become evident on a moment's consideration. It shows that the laborer requires a larger proportion of fatty or carbonaceous food than the idler. Physiologists long regarded the laborer as a foolish fellow when he preferred fat pork to lean beef or chicken for his dinner. Imbued with an erroneous theory which assumed destruction of proteid tissue during work, it was believed that the only method of repairing the proteid waste was to crowd in albuminoid food. The laborer's common sense taught him the true demands of his system long in advance of the physiologist's experiment. The idler and the hod-carrier can maintain their proteid standard on practically the same quantities of nitrogenous food, but the former requires a much smaller proportion of fat and carbo-hydrates. The corollary to be drawn from this is that the easiest way to get rid of an excess of fat is to burn it up by active exercise.

A point of importance to be remembered in this connection is that the amount of fat destructible (oxidizable) by muscular exertion is proportional to the state of muscular development. Hence one of the difficulties of getting rid of an excess of fat in very corpulent individuals by exercise alone lies in the weak muscular development of these individuals.

(To be continued.)

TONSILLITIS.—Let the patient wet his fore-finger and dip it into powdered bicarbonate of sodium. The surface of the tonsil should be rubbed with the end of this finger every five minutes during half an hour, afterwards every hour during the same day. Three applications a day are then sufficient. The author since adopting this treatment has not had to lance a single inflamed tonsil.—*Lyon Médical.*

NOTES FROM SPECIAL CORRESPONDENTS.

PARIS.

KIDNEY Ballottement.—Professor Guyon has indicated a sign that will allow a surgeon to detect an augmentation of the kidney, during its initial period. Hitherto this had not been possible, and, as it is of considerable importance in the early diagnosis of renal complaints, we will state his method. First, as to the method of exploration. M. Guyon remarks that it is usual, in attempting the palpation of the kidney, to get the patient to flex the legs and thighs on the pelvis; but this attitude only seems to make them contract the abdominal muscles, and renders the exploration of the kidney more difficult; so that he finds it preferable to allow the patients to remain quietly in the horizontal position, only taking away the pillows and telling them to breathe slowly and quietly. Then he slips his right hand flat over the posterior region of the kidney, and insinuates the index and medius fingers into the costo-iliac space, and proceeds to raise up this part of the lumbar region by making sharp and repeated pressure. Up to now these shocks have been used to arouse the sensibility of the kidney, which is nearly always manifest in nephritis. However, it is well to commence by searching for pain, as it will be the guide of further exploration. If the pain be severe, the shocks needed to show the *ballottement renal* must be given with great care, so as not to arouse the reflex contraction of muscles, which will interfere with Guyon's sign. The left hand of the operator is now placed flat on the anterior abdominal wall with slight compression, varying according to the case, while the right hand continues the slight shocks until the *ballottement* is felt against the left hand, with something of the same sensation as the abdominal *ballottement* from the fœtus felt in the early months of pregnancy. The pressure of the left hand must not be extreme, because then the kidney will not have room enough, as it were, to move upward and give the sign sought for. When the left kidney is to be examined, owing to its situation, the abdominal hand must be placed higher than on the right side. It must be insinuated under the false ribs a little if possible. This kidney-shock, or *ballottement*, when felt, indicates clearly *an augmentation in the volume of the kidney, and it cannot be obtained when the organ preserves its normal size.*

What is the diagnostic value of this new sign? It is of importance to reveal any slight increase in the size of the kidney *as soon as possible* in all malignant affections of the gland, because its extirpation will be just that much less dangerous and so much more easy as it is least developed. Thus all the kidney-tumors, hydronephroses, renal tuberculosis, and all forms of surgical nephritis will now be better diagnosticated by attempting to get Professor Guyon's sign, and so judging of the slightest augmentation of volume of the kidney, and, as it increases, of the advisability of operation.

Antipyrine.—The application of antipyrine seems to extend day by day. Professor Germain Sée is one of its decided partisans in its use against pain, and goes so far as to count on it in the place of morphine. Its easy solubility allows of its use in subcutaneous injections, and Dr. Sée adopts this form for rheumatic pains in half-gramme doses. It must be stated, however, that at the same time three grammes are given by the mouth, with the result of nearly always calming the pain both in chronic rheumatism and in acute gout.

M. Sée also states that he cured three cases of tic-douloureux, and also cases of painful zona, lumbago, megrim, hepatic colic, nephritic colic, angina pectoris, asthma, and a long list of other troubles, including heart-pains. Professor Sée does not hesitate to conclude that it can entirely replace morphine, and certainly it has not the inconveniences of that drug; but will it always have the fidelity of action that it has against pain? Time alone can tell. At the present moment all the great hospital services are trying antipyrine in all sorts of troubles, so that in a few months its remarkable sedative influence will be investigated enough to enable us to report more fully upon it.

M. Chouppe reports to the Société de Biologie that he had occasion to employ antipyrine in rectal injections to calm uterine colic. In one case a woman was suffering with intense after-birth pains, and an injection containing one gramme of the drug removed the pain. It returned after several hours, but a second injection was given with the result of a definite cure. A second observation was that upon a woman who for several years had violent colic at every menstrual period, which lasted several hours at a time; relief could only be obtained with great difficulty by the use of doses of laudanum or chloral large enough to produce profound sleep. At her last menstrual period, during a most violent attack of pain, one gramme of antipyrine was given by rectal injection, with the result of complete and definite calm being established within a quarter of an hour.

The same author also spoke of the "Reciprocal Action of Antipyrine and Strychnine." He made a number of experiments to see if antipyrine in large doses would modify the form and intensity of strychnine-convulsions, according to a suggestion of Professor Brown-Séquard. He found that the convulsions produced in animals by antipyrine did not at all resemble those of strychnine in three important points: 1st, they were not brought

on by peripheric excitation; 2d, their form was not so tetanic, they consisted of a series of rapid clonic convulsions without any real tetanization of the muscles; 3d, they did not act so much on the muscles of respiration, and this function was not at all suspended with danger of asphyxia, as in strychnine convulsions. Adding the action of strychnine to antipyrine, M. Chouppe injected into the veins of an animal which was already in a state of convulsion from antipyrine a dose of strychnine that should have killed it; but the antipyrine convulsions were simply replaced by strychnine convulsions, and the animal did not die. Then a stronger dose of antipyrine was injected into its veins, which caused the strychnine form to give way to the antipyrine convulsions. The result of various experiments seems to establish that the action of antipyrine to some extent prevents the convulsions of strychnine by reducing the power of the spinal marrow.

Embarras Gastrique.—Professor Peter says he passes a large part of his life talking against bad doctrines and bad medical language, and he considers the above title to be another of the wrong expressions made use of in medicine. Properly, it is intended to express a difficulty in accomplishing a function without any material obstacle,—disordered stomach, in fact; but it almost always is wrongly used, the real trouble being true gastritis. Dr. Peter brings a case before his clinic to prove this fact.

"A young man of 30 years had been examined two years before at a hospital, and the diagnosis given was *embarras gastrique.* This was as false as it often is; for, besides his functional digestive troubles, he had then pain in the epigastric region and slight jaundice, so that it was a real disease, more or less persistent, of the mucous membrane of the stomach, and not a mere disturbance of its function. His history goes to prove this also: he works very hard in a wine-shop, filling bottles, and absorbing the alcoholic vapors, as well as drinking two quarts of wine that he is allowed, not to mention some beer and several doses of bitters, which he takes, so he says, to raise his appetite. His treatment two years ago did him no good, for he continued his habits, with the result that he is now seriously ill. He has cephalalgia, sharp pains in the left hypochondriac region, and extreme prostration with some fever. The digestive difficulty is very marked. We find the spleen very large, with the liver in the same state of congestion, and an impossibility of retaining food of any kind; bleeding from the left nostril exists, and he has petechiæ on his body. So that we have here a patient who is overworked, and indeed poisoned, and all the viscera suffer in consequence. The spleen measures in a vertical direction nineteen centimetres. It is painful upon pressure. This is a fact confirmed by observation in regard to the increase of the spleen in typhoid fever, variola, etc., and in the bad state this man is in he has stored up there in the same way all the products of disintegration. We said that he had 'bleeding from the nose *on the left side*,' and that this fact is a symptom of splenic disease has been handed down to us from Hippocrates, and, passing by Galen, lately our learned friend Professor Verneuil calls our attention to it again. The liver is also increased in size, and the exaggerated increase of the bile-secretion is what Stoll calls 'polycholia:' so that our patient has splenitis and hepatitis, with bilious stools. Our prognosis is very serious.

"What can we do against all these various lesions? In many cases we act through the stomach with our medicines; but here the stomach will not retain anything, let alone drugs, so that we will use revulsion; and you will all testify to the considerable benefit we derive from this therapeutic agency that we have striven for years to insist on the use of, on account of its being so much neglected."

At a later clinic Professor Peter said, "The serious prognosis I gave in regard to the patient you saw last week has happily not been confirmed. I applied at once a large blister to his epigastric region, and the day after his vomiting stopped, so that he could take a little food. Forty-eight hours afterwards a new blister was put on the splenic region, with the result that the pain went away, and to-day the man says he is all right; he has no more pain, and can digest his food. The spleen now measures eleven centimetres instead of nineteen. This man has been in such a low state that I thought at one moment of trying transfusion of blood, and I would have certainly tried subcutaneous injections of ether, which gives such excellent results in serious cases of hemorrhage. But we are relieved of all anxiety by vesication, and I once again, for the thousandth time, call your attention to the marvellous effects of this old remedy of revulsion, which has been allowed to fall into undeserved neglect in these modern days."

Refrigeration of the Healthy Member in Sciatica.—Considerable use is made in France, in the treatment of sciatica, of Professor Debove's method of refrigeration of the line of the painful nerve with chloride of methyl. M. Jacquet now states that immediate cessation of pain can be obtained by refrigeration *of the healthy leg* (!) In ten cases so treated he obtained six cures, which lasted for several days only, however, so that the duration of cessation of pain is much less than by the usual method, and the effect seems to diminish by use. M. Dumontpallier said that these results are in accord with those that he had observed, and he thought he could explain them by admitting that all these actions are produced because the pain has a central ori-

gin. What happens in the case of the sciatic nerve is analogous to what is seen in the stitch in the side in pleurisy and pneumonia, for instance, which will often give way under the influence of a blister *applied on the healthy side.*

M. Ch. Richet said that when one hand was exposed to cold the other one felt in a short time a spasm similar to that felt in the opposite hand. This is a reflex action of the same order, as is well known.

Restoration of the Lower Jaw with the Bone of a Calf.—Professor Richelot read a report of this case at the Société de Chirurgie. It was a young girl of 16, who had been subject to frequent attacks of hysteria, and came to Dr. Richelot for a congenital malformation of the jaws which made mastication almost impossible. The lower jaw was most affected, as its left side was thrown downward and outward, and had a curve much longer than the opposite side, so that proper articulation between the teeth was prevented. On the right side, with the aid of rubber attachment (put on by a dentist), she could triturate slightly some forms of food. Dr. Richelot concluded to make an orthopedic resection of the lower jaw, and on the 25th of July last year he took away, by the aid of Liston's pincers and a perforator, about two centimetres of the bone at the level of the left bicuspid teeth, making a suture with silver wire and exact coaptation of the fragment, adapting the canine-tooth part with the molar portion. This operation was made by the mouth to prevent any exterior cicatrix, but, owing to the insubordination of the patient and the hysterical attacks she was subject to, bony reunion did not take place, and she left the hospital with a pseudarthrosis. Some two months later M. Routier received the girl at the Laennec Hospital, and undertook to cure the pseudarthrosis. At first he thought of using an ivory pin, but after reflection he concluded to use a piece of calf's bone taken from an animal lately killed. The operation was done the 13th of January, 1887, by an incision made on the inferior border of the inferior maxillary, destruction of the fibrous tissue, scraping of the fragments, and introduction of the portion of calf's bone, which secured perfect immobility. The reunion was made without much difficulty, and the bony consolidation followed. The patient was seen the 12th of March last, and was quite well, being even cured of her hysteria, and able to use her jaws quite well, although there is still a slight defect in the articulation of the teeth. M. Richelot added that he was disposed to try putting a slip of animal bone between the fragments in any such resection again, as he saw the perfect consolidation that was obtained after his failure by suture, perforation, and the usual method. The sensibility of the inferior dental nerve was not studied, but there is nothing abnormal at present, and the sensibility of the lips was perfect.

Prophylaxis of Transmissible Diseases.—Dr. Martin, in charge of Professor Graucher's service, makes some good remarks in regard to the above subject. Hygiene, he says, is of the utmost importance, and whether we believe or not in the importance of the *rôle* acted by the micro-organisms in the pathogeny of disease, hygiene can act all the same. It is enough to know that some sort of contagion exists, and that it is transmissible, in order to have good reason to prevent its transmission. Most of us know enough to make a prescription when we see a disease, but perhaps it would be better for us if our clients did as the Chinese, and pay as long as they are kept well. The means to be used to prevent propagation of many affections are three in number,—isolation, disinfection, and official information: there is a fourth, that is vaccination, that is good for a single disease, and there is hope that it may be extended to others. Isolation is certainly of very great importance, and not carried out as it should be. All persons apt to contract the disease must be rigidly kept away from the sick-room, particularly children; those who nurse the patient must be kept from the rest of the family, they must not eat or drink in the sick-room, and they must be careful to wash their hands with antiseptic solutions constantly. All curtains, carpets, and hangings must be taken away from the room, and the bed placed in the middle of it; the windows must be opened twice daily, covering the patient well. The time of isolation varies for different maladies, but in a general way twenty-five days will do for varicella and mumps, forty days for measles, scarlatina, and small-pox. In every case the patient must be well enough to have had a bath before seeing any one socially. *Disinfection* is the second means to be used, and Vallin understands by this the complete destruction of all the emanations from the patient, if they smell or not. The number of chemical products used should be small, and fire with steam be added. Corrosive sublimate in one-one-thousandth solution should be preferred, but it has a number of difficulties: one is, it is dear; another, it looks like water; again, it attacks domestic utensils, and cannot be used in strong solutions. So that carbolic acid is best for general use: owing to its characteristic odor, it will not be taken for anything else. All fæcal matters, urine, vomited matters, etc., should be disinfected at once, and the mixture then placed in a wooden box filled with earth, and not thrown into any stream or the usual closets. When any article used is not of much value, it should be destroyed by fire. Articles of value should be steamed under pressure. If this cannot be done, do not give the linen to the usual washwoman until it has been kept

for a considerable time in a solution of hot antiseptic fluid. Finally, it is the duty of the doctor to inform the city authorities; and while the law does not compel him to do so, except in cases of cholera, pest, or yellow fever, it is held by Professor Brouardel that in doing so we do not give up the rights of the *secret médical*, but we assist the city to use efficacious measures in stamping out the first signs of an epidemic. The law of silence only applies to venereal diseases or those that may be hereditary: so aid your patients by government or city prophylactic measures when you can. Dr. Martin closes by giving a table that sums up some very interesting facts of difference in mortality, births, etc., in countries like England, Germany, and France, where efforts are made to sterilize soil and localities by proper means leading to general and local salubrity and causing a diminution of epidemics. We give a few of the figures (the most striking):

	Births per 1000.	Deaths per 1000.	Excess of births over deaths.	Fecundity of women per 1000.
Germany.....	37.2	25.7	11.5	151
Italy.....	36.9	27.9	9	148
Switzerland....	35.9	25.7	10.2	114
England.....	34.2	19.9	14.3	130
France.....	24.9	22.6	2.2	99

So that while the mortality in France is not elevated, the excess of births over deaths is very small and the fecundity of the women low.

THOMAS LINN, M.D.

PARIS, August 1, 1887.

PROCEEDINGS OF SOCIETIES.

THE ASSOCIATION OF AMERICAN PHYSICIANS.

(Continued from page 775.)

CASES OF SEWER-GAS POISONING. BY DR. HENRY HUN, OF ALBANY.

THE author reported in detail the histories of twenty-nine cases coming under his observation, in which various diseases appeared to have been due to the inhalation of sewer-gas. He thought it probable that the following diseases may result from sewer-gas poisoning: vomiting and purging, separately or combined, general debility, fever, sore throat of a diphtheritic type, neuralgia and perhaps also myelitis of the anterior horns. These conditions are frequently combined. Fever is frequently associated with the other symptoms. There is one group of symptoms which is almost always present, that is, loss of appetite, extreme prostration, and pain in the head. When this occurs as a chronic condition, we are justified in suspecting that the patient is suffering from sewer-gas poisoning.

Israel T. Dana, M.D., of Portland, Maine, reported a case of aneurism of the abdominal aorta cured by rest, restricted diet, and potassium iodide.

DISCUSSION ON HEMORRHAGIC INFARCTION.

At the evening session the discussion on hemorrhagic infarction was, by appointment, opened by Dr. W. W. Welch, of Baltimore.

The author first referred to the different theories which had been advanced to explain the occurrence of hemorrhagic infarctions. These are (1) changes in the walls of the artery obstructed; (2) the increased pressure with which the blood is sent in from the collateral circulation; and (3) a regurgitant flow of blood from the veins. Numerous experiments had been performed by the author, with the assistance of Dr. Mall, of Johns Hopkins University, to determine which of these theories was the correct one. Hemorrhagic infarctions were produced in the intestines of dogs, and the method of experimentation was given in detail. He presented as the result of his studies the following conclusions:

1. The blood which produces hemorrhagic infarctions comes chiefly, if not exclusively, from the collateral vessels.

2. Hemorrhagic infarctions in the intestine cannot take place merely from a reflux of blood from the veins.

3. The blood-pressure is very low in the region where hemorrhagic infarction occurs, in consequence of occlusion of the main artery.

4. A certain degree of force of the collateral circulation is required to produce a hemorrhagic infarction.

5. No positive proof exists that a change in the vascular walls is essential to the production of a hemorrhagic infarction.

6. The hemorrhage occurs by diapedesis.

7. Where hemorrhagic infarction has taken place the large and small veins are widely dilated with blood, and the arteries contain a smaller quantity of blood than normal. There is stasis in many of the veins and capillaries.

Dr. William Osler, of Philadelphia, the conferee, referred to the

CLINICAL ASPECTS OF HEMORRHAGIC INFARCTION.

Among other cases coming under his observation, he reported the following:

J. M., age 20, admitted to the Philadelphia Hospital October 10, 1886. He had never had syphilis, and was a healthy-looking man. He presented a clear history of typhoid fever with a sickness of six weeks two years previously. His present illness began with diarrhœa one week before admission. For two days he had attacks of bleeding at the nose. There was temperature of 102°, with pain in the abdomen. There was no cardiac mur-

mur, and examination of the lungs gave negative results. The splenic dulness was increased. By October 15 the temperature had reached 103°. There was almost constant delirium. There was some diarrhœa. Coldness of the feet appeared, and continued to increase in degree and extended up the leg. The legs became livid, and no pulsation could be detected in the femoral and popliteal vessels. The patient died on the 17th.

It was supposed that there was thrombosis of the iliac veins with gangrene of the legs, which is one of the rare sequences of typhoid fever. At the autopsy it was found that the lower portion of the abdominal aorta and also the two iliac arteries were plugged with thrombi. There was general peritonitis. The right kidney presented a red-brown infarction. There were no ulcerations in the bowel; no endocarditis. The lungs were normal. There was an infarction in the spleen. During life the blood was examined for microbes, but none were found. After death microbes were found in the spleen.

Hemorrhagic infarction of the liver, under ordinary circumstances, is impossible. A. B., a hard drinker, was admitted to the hospital September 27. His illness began in the previous June, with vomiting and swelling of the abdomen. The dropsy steadily increased. He died two days after admission. At the autopsy a large amount of fluid was found in the peritoneal cavity. There was nothing special found in the heart or lungs. The liver was remarkably cirrhotic. Through the right half of the right lobe there were scattered numerous reddish-brown areas. The walls of the portal vein were thickened, and a large brown thrombus occupied the upper portion of its trunk. The branches passing to the right lobe were filled with clots. The hepatic artery and vein were normal. In this case the hemorrhagic infarctions were in all probability due to the cirrhosis of the liver, which had caused more or less obstruction of the branches of the hepatic artery. The only other case of infarction of the liver which the speaker had been able to find was one reported by Recklinghausen.

In the intestine hemorrhagic infarction is met with in two forms: one involving the mucosa, the other affecting the entire gut. The former not infrequently ends in ulcerative necrosis. The latter form of hemorrhagic infarction is not common in man; in the horse it is frequently seen resulting from thrombi formed from verminous aneurisms of the mesenteric and its branches. This is a common cause of the severe and fatal colic so frequently seen in these animals. In conclusion, the speaker referred to the fact that in the lung it was not uncommon to have a vessel blocked without the production of an infarction. An occasional cause of thrombotic infarction is local disease of the pulmonary artery. It sometimes results from the endarteritis induced by beginning tubercular processes.

In the discussion on these papers Dr. Reginald H. Fitz, of Boston, reported a case which in his experience was quite unique. It was that of an elderly man with globular thrombi in the left ventricle. Emboli were transferred to the splenic artery, but at first were not sufficient to completely occlude the artery. The spleen became enlarged, and thrombi formed in the splenic veins, and from this point the thrombus extended into the superior mesenteric vein. As a consequence, hemorrhagic infarction occurred in the intestines.

Second Day.—Morning Session.

BERGEON'S METHOD OF TREATING PHTHISIS. BY DR. E. T. BRUEN, OF PHILADELPHIA.

With reference to the effect of the injections of sulphuretted hydrogen on the bacillus tuberculosis, Bergeon does not claim that their number has been reduced in any considerable proportion of cases. In the treatment of phthisis there are three main indications. The first is to secure some agent which will act upon the cause of the disease. Climate, diet, and hygiene serve to modify the predisposing causes; but if the bacillus be the real cause of the disease, we lack any agent which will destroy it. The second indication is to prevent the destruction of the tissues, for it is found that as the vitality is increased the number of organisms in the sputa is diminished. The third indication is to control special symptoms which present themselves. Since February last I have employed Bergeon's method of treatment in sixty-one cases. Forty-four of these cases have been benefited, but of these only three appeared to regain full health. Two of these were cases of incipient phthisis, with apparent consolidation of the apex of the right lung. In one of these cases the bacillus tuberculosis was not found, though five examinations were made. In the other case the bacillus was found. In these cases the apparent recovery has been associated with a decided increase in weight. He believes, however, that the disease is simply latent. The third case was one of bronchopneumonia. In all the other cases the lesions were more or less advanced, with the presence of cavities and profuse expectoration. In the fifteen cases in which a negative result was obtained, the treatment in some was followed by temporary benefit. The good results have consisted in lessening of the expectoration, diminution of the cough, lowering of the temperature, and suspending of the night-sweats. In most of the cases there was a diminution of from fifteen to twenty beats in the pulse, and a diminution of half a degree in the temperature during the administration of the gas. Even in those cases

which were benefited, and in which the temperature had been brought to normal, there would be, during the progress of the treatment, occasional outbreaks, with a return of the fever and the other symptoms. These, however, disappeared under a continuance of the injections. In order to determine the effect of the treatment on the bacilli, Dr. E. O. Shakespeare made a number of examinations during the progress of the cases. There has been no diminution in the number of the bacilli. It was, however, thought that in those cases where the treatment had been continued for some time, the reaction of the bacilli to the staining-fluid was less marked.

Two cases have died. In one of these an autopsy was made. This case had been under treatment for two months. The walls of the cavity were moderately smooth and firm, but there was no tendency to cicatrization. The results were of a decidedly negative character.

In most of the cases a solution made by the addition of five grains each of sodium sulphide and sodium chloride to a pint and a half of water was the solution employed. In some cases the strength of the solution had been gradually increased, but where no benefit had been obtained from the weak solution the stronger solution did not act with any better results. The quantity of gas employed at each injection has been about one gallon. If there is pain, the strength of the solution may be decreased or a smaller quantity of the gas may be given. At least an hour should be consumed in each administration of the gas. The patient should rest quietly in bed an additional half-hour until the gas has been absorbed. The admission of atmospheric air should be avoided.

In cases of diarrhœa his experience had been unfavorable, except where the gas was given in very small quantities. Chronic peritonitis is a contra-indication to the use of this plan of treatment. In about one case in every ten he had observed the reaction of sulphuretted hydrogen when a paper treated with acetate of lead had been applied to the mouth. This would indicate that a very small amount of the gas reaches the lung, and that the efficiency of the treatment does not depend upon the use of a strong solution. In no case have any injurious results been seen. In some cases where strong solutions were employed, the appetite and strength seemed to be impaired.

Bergeon's method is chiefly valuable in those cases attended with bronchial catarrh. He had had very little good effect in those cases where there was thickening of the lung without much catarrh. The speaker feared that the trouble and detail necessary to the successful application of this method, and the limitations of its power, would cause it to be set aside for other therapeutic measures.

(To be continued.)

BRITISH MEDICAL ASSOCIATION.

Fifty-Fifth Annual Meeting, at Dublin, August 2–5, 1887.

(Continued from page 779.)

T. GRAINGER STEWART, M.D., Professor of the Practice of Medicine in the University of Edinburgh, read, in the Section on the Practice of Medicine, a paper on

The Incidence of Albuminuria among the Sick.

Supplementing the inquiry as to the incidence of albumen in the presumably healthy, reported to the Royal Society of Edinburgh, the author had made a series of observations on various groups of patients to which he had access as to its occurrence among them. These patients consisted of one hundred and fifty consecutive private patients, one hundred and fifty consecutive cases from the wards of the Royal Infirmary, one hundred consecutive out-patients, fifty cases from the wards of the Hospital for Sick Children, forty cases from a ward devoted mainly to the treatment of cases suffering with alcoholism, fifty patients from the Fever Hospital, and twenty-five cases of women from the Maternity Hospital. In each case the urine was examined with nitric acid, picric acid, and Fehling's solution.

The author then presented a series of tables.

Table I. showed that, among the private patients, thirty-six, or twenty-four per cent., showed albumen; among the in-door patients of the Infirmary, nineteen per cent.; among the sick children, seven, or fourteen per cent.; among the fever cases, thirty-three, or sixty-six per cent.; among the maternity cases, eighteen, or seventy-two per cent.

Table II. dealt with the causes of the incidence of the albuminuria in the one hundred and fifty private cases. Thirteen out of the thirty-six were due to Bright's disease, one febrile, two vascular, three alimentary, two glycosuric, six functional, eight clearly accidental, one probably accidental.

Table III. dealt with the probable causes in the one hundred and fifty in-door Infirmary cases. Seventy-four cases of albuminuria were met with in the wards; twenty-six were from Bright's disease, five febrile cases, sixteen vascular, six alimentary, five nervous, three glycosuric, five accidental cases.

Table IV. dealt with the probable cause in one hundred out-door Infirmary cases. Of nineteen showing albuminuria, eight were from Bright's disease, three as probably Bright's disease, five vascular, one alimentary, one glycosuric, one probably accidental.

Table V. dealt with the cause in the fifty children. Bright's disease occurred in one case out of the seven, probably Bright's disease two, febrile three, nervous one.

Table VI. showed the results obtained as to

the occurrence of albuminuria among fifty fever cases in the Edinburgh Fever Hospital. Of these, eighteen, or thirty-six per cent., showed albumen with nitric acid, and thirty-three, or sixty-six per cent., with picric acid. Taking the thirty-six cases of scarlet fever separately, we find that thirteen, or 34.2 per cent., showed albumen with nitric acid, and twenty-three, or 60.53 per cent., with picric acid.

Table VII. showed the results in forty cases of alcoholism, either acute or chronic, treated in the Royal Infirmary. The specimens were obtained on the morning after their admission. One would naturally expect a large proportion of albuminurics among such patients, and no doubt the percentage is high. Still, we must remember that the class of patients of which this group is composed is most closely related to that of the soldiers,—that is, the conditions of life, such as heavy bodily labor, etc., which in soldiers conduce to make albuminuria frequent are also operative here. Still, the percentage of albuminurics is distinctly higher than it is among the soldiers, and indeed more closely corresponds with the statistics obtained among the in-door Infirmary patients. While, therefore, the albuminuria is probably mainly to be ascribed not to alcohol, but to the conditions of life, the alcohol must be credited with the increase over what we would expect in healthy individuals of the same class. This may be partly a direct effect of the alcohol, but doubtless the indirect influences as regards inferior diet, and bad hygienic conditions, which intemperance involves, are also factors in the case.

The next question is, How are we to explain the albuminuria in these various conditions? He did not attempt to do this until he had gone over the results of experiments made by different observers with the view of clearing up the question. Certain inferences seem to be warranted from the facts just stated:

1. That in health, so in disease, albuminuria is more common than is generally supposed. 2. That it is more common among patients of adult age than among children. 3. That cases of Bright's disease do not account for one-half the cases of albuminuria met with in practice. 4. That they account for more than any other individual cause. 5. That next to them rank the changes in the renal circulation due to cardiac and other maladies, and accidental cases due to the admixture of blood, pus, etc., with the urine. 6. That, so far as this series of observations shows, cyclical, dietetic, and simple persistent albuminurias are rare. 7. That while in Bright's disease, especially the waxy and cirrhotic forms, the quantity of albumen is at first so slight as to be only shown by picric acid, it yet is more abundant than in albuminuria due to other causes. 8. That in the digestive and nervous cases, and those due to high temperature, the quantity is often so small as only to be discovered by picric acid.

We now turn to note the results of his observations as to the occurrence of peptones in the series of cases examined.

Table VIII. shows incidence of peptonuria in the series of cases examined. It thus appears that only four out of one hundred and fifty patients showed this condition. These cases were (1) a case of cirrhotic Bright's disease with gout and dyspepsia, (2) a case of cirrhotic Bright with heart-disease, (3) a case of rupture of the kidney, and (4) a case of acute rheumatism. The urines were all albuminous. Of one hundred and fifty in-door patients at the Infirmary, sixteen gave the reaction. These were a case of dyspepsia, a case of malignant disease of the liver, stomach, lungs, etc., a case of heart-disease, two of phthisis (one of them syphilitic), a case of pleurisy, nine cases of Bright's disease, and a case of acute rheumatism. Except in the case of dyspepsia, all the urines contained albumen as well.

In the Obstetrical Section, John W. Byers, M.D., of Belfast, read a communication entitled

THE PREVENTION OF PUERPERAL FEVER IN PRIVATE PRACTICE.

The paper gave some statistics showing the great mortality from puerperal fever; but the writer said it was impossible to take measures for its prevention until we had clear ideas as to its causation. After he had discussed various views, he gave his reasons for believing that it was blood-poisoning analogous to septicæmia met with in surgical practice. Taking this view, he believed there were three principal sources by which the poison might be introduced into the system of a lying-in woman:

1st. Sewer-gas, as in unhealthy homes.
2d. The nurse.
3d. The accoucheur.

Under the first head he recommended the inspection of a house before the confinement took place, to see if it was in a proper sanitary condition. He gave rules for the guidance of the practitioner and nurse. He advised the strictest cleanliness, thorough washing of the hands in an antiseptic solution (one to one thousand) of perchloride of mercury, few examinations, a proper management of the third stage, the keeping of the patient on her back for a time after confinement, and the use of antiseptic douches.

He drew attention to the cases in which a decomposing blood-clot or placenta might be the source of the disease, but believed that even in these cases the decomposing mass in the uterus only acted as a nidus in which the poison from without found suitable soil.

He discussed the question of how a physician should act with reference to obstetric practice if he came across cases of scarlatina

or foul surgical wounds, and said he believed by bathing, changing the clothes, and strict antiseptic washings he need not fear to attend a confinement. He said if those who attended women in labor would act as if each one were likely to develop septicæmia, they would take thorough antiseptic precautions, and thus such cases would be reduced to a minimum.

The whole essence of the prevention of puerperal fever was—

1st. To prevent the poison entering the system.

2d. To destroy the poison, if it should get into the genital tract of a patient, before it entered her tissues and blood.

Dr. Robert Barnes read a paper on

THE CAUSES, INTERNAL AND EXTERNAL, OF PUERPERAL FEVER.

A clear understanding of the nature and causes of puerperal fever is the natural antecedent of a rational system of prevention. What is puerperal fever? What are its factors and constituents?

There is not one cause, but a plexus of causes. Failure to comprehend this truth is especially conspicuous in the German works. By a short and arbitrary process of synthesis this school formulated the dogma that puerperal fever is puerperal septicæmia, the result of septic infection from the genital canal, thus overlooking important constituent factors. Starting from the state of gestation, we find it especially marked by exalted nervous and vascular tension. An acute process of building up is going on. Parturition puts to the test every tissue, every function. We have three combined sources of altered blood: first, that derived from gestation; second, that arising during labor; third, that arising from the disintegration of the superfluous tissues built up during gestation. Labor completed, a process is started the very reverse of that which prevails in the gravida. The high vascular tension subsides; the tide turns; the blood is invaded by waste stuff which has to be cast out of the body. This is done mainly by the excreting glands: the liver, kidneys, skin, intestines, lungs, are all called upon for active work. They must discharge the waste stuff as fast as it is received into the circulation. If this waste stuff be not duly excreted, it accumulates and forms foyers of poison. Cases of thrombosis and phlegmasia dolens are mainly the expression of the loss of balance of disintegration and elimination.

This is the simplest, the fundamental form of puerperal fever; but it is the compound of three forms of altered blood: 1, the blood of the gravida; 2, the blood of the parturient; 3, the blood of the puerpera. This is purely autogenetic. I call it endosepsis. The conditions arise entirely within the patient's system.

Another group of symptoms which disturb the orderly course of the puerperal state are influences which retard the secretions and excretions, as chills, malarious conditions, errors of diet (among which insufficient diet is one), bad hygienic surroundings, and emotions.

Meteorology controls these secretions largely, and I believe has no little effect on the frequency of puerperal fever. I show here a number of charts. The first gives, according to the Royal Meteorological Transactions, the general temperature and rainfall for thirty years,—1844 to 1874; the second shows the deaths from scarlatina, fevers, erysipelas, and puerperal fevers for the same period; the third shows the total births and deaths for the same period. The second series I have constructed on the same plan. It consists of the history for ten years,—1875 to 1884. The months and weeks of each year are cast together, so that January consists of thirty weeks, corresponding. In the second series we get the curve for the following ten years. The separation of ten years can be compared with the preceding thirty years. The general similarity of the curves is remarkable. It affords strong evidence of the universal prevalence of the causes. The tables exhibited showed the relation of the seasons and atmospheric conditions to fevers, puerperal and others, and illustrated a law which has been widely recognized, that zymotics are most fatal in the winter. This I believe is true. The damp from the cellars, charged it may be with foul water, but noxious in any case, is sure to be sucked up into the house, and seeks by preference the warmest spot. This is the lying-in room, the heat acting as a relative vacuum in the highly-heated and poorly-ventilated room. This source of danger is distinct from the ordinary drains or sewerage of the house: either may act alone, or they may act together, intensifying the danger.

This damp foul air, drawn into the lying-in room, may be contaminated with specific poisons, as of typhoid, scarlatina, or of erysipelas; but the contamination is not necessary to work mischief in the puerpera. This evil, that the lying-in room is chiefly supplied by air from the lower regions in winter, need hardly be felt in summer, as fresh air is brought directly from the outside. Pressure then works against the up-current; pure air is gained, foul air is repelled. [Here the author showed a model house to prevent this suction of foul air.] During the past winter, an unusually inclement one, I have been called to attend an unusual number of cases of chills and fever. I rarely failed to find sufficient cause for the trouble in the sanitation. I would enforce emphatically the old rule to move the patient to another room, preferably the drawing-room, which is generally the healthiest, most airy in the house.

Sewer-gas, if not carrying zymotic germs, may cause fever. The poison, however, must be kept up. Hence the advantage gained from moving the patient out of her surrounding influences.

As to decomposing matter in the genital tract, we should first close the gates against the entry of the enemy by securing firm contraction of the uterus, and prevent the suction-action of the abdomen. The next step is to give ecbolics, as quinia, cinnamon, nux vomica, ergot, and digitalis. Some combination of these medicines should be given in every labor. We should then eject the enemy and prevent the gathering force by washing out the uterus. Uterine irrigation is especially useful in the autogenetic forms in which noxious matter is taken up from the genital tract. It is not frequently necessary in the heterogenetic or zymotic forms.

The women of the laboring classes go through child-bearing more safely than their pampered and delicate sisters. It is because they are not subjected to and enfeebled by excessive cultivation of the emotional and intellectual elements. The daughter of luxury never knows a day's labor till she is taken in labor, while the woman of the working-classes labors every day.

Lactation is evidence of glandular capacity; failure to produce milk, presumptive evidence of glandular incapacity. Fever prevents or diminishes secretion. In such cases, at least, failure to secrete milk is a primary, not a secondary condition. Fever declares itself because the glands cannot do their duty.

From what precedes, it follows that to insure the fullest security to the puerpera, to put her in the best position to go through the transition from the puerperal to the ordinary state, we must do what we can to keep every organ, including the glandular system, in good working order, and to guard against all known agencies which may disturb the healthy action of the organs.

The relations of meteorology to puerperal fever is a point to which I wish to call especial attention. We see by the tables that puerperal fever, like other fevers, prevails most fatally in winter. To what extent can we control the pernicious influences of winter weather? Domestic meteorology is greatly under our control. We must inquire what are the external conditions which favor correction. The excretory force of the lungs is increased in value in the puerpera. Under ordinary conditions in the non-pregnant they gave off watery vapor, carbonic acid, sometimes ammonia and a minute quantity of organic matter. It is certain the capacity of the lungs increases after labor, so it is probable the quantity of carbonic acid exhaled is increased, and certain the exhalations of organic matter increase. The "gravis odor puerperii" is partly due to this increase. In some cases, though verging closely on antisepsis, the lochial discharge is absorbed. In these I have noted a sallow aspect and a degree of febridity, and I have taken this as an indication for irrigating the cavity of the uterus.

To get rid of the excess of aqueous vapors of carbonic acid and organic excreta by the lungs and skin, a constant supply of pure air is required. It must be dry and comparatively rare. There are two principal modes by which gaseous matter including germs or bacteria is carried: 1, by diffusions; 2, by currents of air. Active diffusions are especially valuable so long as they bring nothing noxious to the patient; but if foul matter comes with the air it will tell against the case. Hence the great importance of a supply of pure air and the removal of all impediments to free circulation. I am disposed to believe the most injurious of all meteorological conditions is damp in the form of saturated air, fog and mist. Wind, rain, and sun are the great purifiers of the atmosphere. Movement of the air promotes diffusion, sunshine works chemical changes, and the rain scours or washes away impurities. All of this, of course, is familiar knowledge; but it is not superfluous to ask if it is fairly applied to the regulation of the sick-room. Let us consider how far we may bring the meteorology of the bedroom to the conditions of healthy external meteorology. 1. Let the lying-in chamber face the south. To combat external damp a fire is best in an open fireplace; the open grate serves as a ventilator to carry off impurities. Among the abominations of self-styled sanitary engineers, I feel called upon to denounce heating by hot-water pipes. They are too often effective means for the culture and diffusion of noxious germs. It is possible also to wash the air by air admitted by a Dobin's tube, by a shower of water falling into the tubes.

The modification of the air by chemical agents, with the mechanical addition of vapors and gases possessing medical virtues.

In the early morning hours, between two and four o'clock, is the time when the wind is the lowest and the meteorological conditions the worst. At this time the ventilation of the room should be attended to with greater care than usual.

All precautions resolve themselves into one word,—cleanliness. All antiseptic precautions are but a part of the general scheme.

The meteorological conditions are of primary importance, though far from underestimating other causes.

Whilst protecting the citadel from the foe without, we should take care that mutiny does not break out within.

(To be continued.)

THE MANAGERS OF THE PENNSYLVANIA HOSPITAL gave a reception on August 31 to visiting physicians on their way to attend the International Medical Congress.

NEW REMEDIES AND CLINICAL NOTES.

IMMUNITY BY INJECTION OF CHEMICAL BODIES.—Dr. L. C. Wooldridge recently communicated to the Royal Society a method by which he had been able to protect rabbits from anthrax, which is of considerable interest in connection with the general question of the nature of protection in this and other diseases depending on micro-organisms. The method consists in cultivating the anthrax-bacillus in an alkaline solution of a peculiar proteid body which can be obtained from the testis and thymus gland. The growth is not abundant, and after two days at 37° C. it is removed from the culture-fluid by filtration. A small quantity of filtered liquid is injected into the circulation of a rabbit, and the animal can then withstand the inoculation of extremely virulent anthrax blood. The bacillus itself grown in this peculiar culture-fluid has no protective influence: it either kills or it has no effect. The result is extremely curious, for hitherto protection against zymotic disease has been effected by the communication to the animal of a modified form of the disease against which protection is sought. In Dr. Wooldridge's experiments the protection must be produced by some chemical body the product of the activity of the bacillus. The observation belongs to a new order of facts, and appears to fall in with M. Pasteur's theory as to the method in which immunity to hydrophobia is produced by inoculation of the spinal cord of rabid rabbits. Both find some support in Professor Cash's experiments with perchloride of mercury, in which it was shown that after animals had taken a sufficient quantity of the drug they were no longer liable to anthrax.

CORROSIVE SUBLIMATE IN INTRA-UTERINE IRRIGATION.—Dr. Braun, from recent observations, has arrived at the following conclusions concerning the use of corrosive sublimate in irrigation of the uterus and vagina: 1. Vaginal or intra-uterine irrigation is frequently followed by absorption of the injected liquid. 2. When this occurs, mercury is quickly detected in the fæces. 3. If the return of the injected liquid be in any way prevented, absorption occurs rapidly. 4. The one in one thousand solution of sublimate should be used only in serious cases, such as tympanites of the uterus, putrefaction of the fœtus in the uterine cavity, or septic puerperal fever. The injection should not occupy more than a minute in the performance, and should be followed by a copious injection of distilled water. 4. The four in one thousand solution should be injected only in cases of expulsion of a macerated fœtus or in endometritis consecutive to the expulsion of the fœtus in premature delivery. 6. This solution may be of service in puerperal endometritis accompanied by a fetid vaginal discharge; in these cases irrigation should be followed by an injection of pure water. 7. Irrigation should be performed only by a medical man. 8. Irrigation with corrosive sublimate should seldom be employed in women suffering from extensive wounds of the vulva, in those who have been taking mercurial preparations, in cases of atony of the uterus, in anæmic women, or in patients suffering with disease of the kidneys.

ACUTE CHOREA TREATED WITH PHYSOSTIGMINE.—By administering the remedy subcutaneously, Riess claims that the duration of the disease is shortened to about a fortnight, and that forty out of forty-four cases were treated successfully with physostigmine, the remaining four being of the very severe form.

MISCELLANY.

THE AMERICAN DERMATOLOGICAL ASSOCIATION held its eleventh annual meeting in Baltimore, August 31 and September 1 and 2, at the hall of the Medical and Chirurgical Society of the State of Maryland. Abstracts of the principal papers will be given hereafter.

COOPER HOSPITAL OF CAMDEN.—The staff of the new institution consists of—Surgeons, Drs. Dowling Benjamin, O. B. Gross, J. F. Walsh, and E. L. B. Godfrey; Physicians, Drs. J. McCray, D. P. Pancoast, W. A. Davis, and H. Genet Taylor; Resident Physician, Dr. Harry Jarrett; Pathologist, Dr. Joseph H. Wills.

NOTES AND QUERIES.

OBITUARY.

NATHANIEL ARCHER RANDOLPH, M.D., Professor of Hygiene in the University of Pennsylvania, was accidentally drowned while bathing, August 21, at Longport, near Atlantic City, New Jersey. It is believed that death was really due to a heart-affection from which he had been suffering for some years, and that sudden cardiac failure resulted from violent muscular efforts while struggling in the water to escape from being carried out by the undertow. Dr. Randolph was born at Chadd's Ford, in this State, November 7, 1858, and was a graduate of Cornell University and the Medical Department of the University of Pennsylvania. He was a member of the College of Physicians of Philadelphia and of other societies, and had been for a short time one of the editors of the *Medical and Surgical Reporter*. He especially interested himself in the study of nutrition, and had contributed several papers on the relative value of foods.

E. W. GERMER, M.D., ex-President of the State Board of Health, died at Erie, August 22, at the age of fifty-six. He was born in Altsbrisach, Baden, Germany, and graduated in medicine at the Vienna University in 1859. During the Baden revolution he was among the insurgents, and was severely wounded. Coming to Erie, he engaged in the practice of medicine. When the smallpox scourge developed in Erie, he was nurse, physician, undertaker, and clergyman. He was appointed on the State Board of Health by Governor Pattison, and was President of that body last year. He was a member of the National Board of Health, and at the last meeting of the State Board was elected to represent Pennsylvania at the International Sanitary Convention at Berlin next month. While on the State Board, he was on the committee which investigated and located the cause of fever-contagions at Plymouth and Pittsburg. It was through his efforts that the grave of General Anthony Wayne was finally located.

OFFICIAL LIST

OF CHANGES IN THE STATIONS AND DUTIES OF OFFICERS SERVING IN THE MEDICAL DEPARTMENT U.S. ARMY FROM JULY 31, 1887, TO AUGUST 27, 1887.

COLONEL CHARLES SUTHERLAND, SURGEON.—Leave of absence extended one month. S. O. 174, A. G. O., July 29, 1887.

MAJOR J. C. MCKEE, SURGEON.—Ordered for duty at Watertown Arsenal, Massachusetts. S. O. 181, A. G. O., August 6, 1887.

MAJOR B. E. FRYER, SURGEON.—Relieved from further duty at Fort Lowell, Arizona. S. O. 176, A. G. O., August 1, 1887.

MAJOR HARVEY E. BROWN, SURGEON.—Relieved from duty in Department of Missouri, and ordered to Jackson Barracks, Louisiana, for duty at that post. S. O. 174, A. G. O., July 29, 1887.

MAJOR H. E. BROWN, SURGEON.—Sick-leave extended to August 6, 1887, on account of sickness. S. O. 184, A. G. O., August 10, 1887.

MAJOR P. J. A. CLEARY, SURGEON.—Ordered to Fort McDowell, Arizona Territory, instead of Fort Assiniboine, Montana Territory. S. O. 180, A. G. O., August 5, 1887.

CAPTAIN JOHN M. DICKSON, ASSISTANT-SURGEON.—Died August 8, 1887. (Station, Fort Mason, California.)

CAPTAIN JOHN DE B. W. GARDINER, ASSISTANT-SURGEON.—Granted leave of absence for one year, on surgeon's certificate of disability. S. O. 177, A. G. O., August 2, 1887.

CAPTAIN J. C. MERRILL, ASSISTANT-SURGEON.—Ordered from Fort Klamath, Oregon, to Watervliet Arsenal, New York. S. O. 181, A. G. O., August 6, 1887.

FIRST-LIEUTENANT WILLIAM E. HOPKINS, ASSISTANT-SURGEON.—Ordered from Angel Island, California, to Fort Mason, California. S. O. 184, A. G. O., August 10, 1887.

FIRST-LIEUTENANT C. N. B. MACAULEY, ASSISTANT-SURGEON.—Promoted to be Assistant-Surgeon with the rank of Captain, by operation of law, August 10, 1887.

FIRST-LIEUTENANT J. E. PILCHER, ASSISTANT-SURGEON.—Ordered from Fort Monroe, Virginia, to Fort Wood, New York Harbor. S. O. 180, A. G. O., August 5, 1887.

FIRST-LIEUTENANT C. L. G. ANDERSON, ASSISTANT-SURGEON. (Station, Whipple Barracks, Arizona Territory.)—Ordered to Fort McDowell, Arizona Territory. S. O. 81, Department of Arizona, August 3, 1887.

FIRST-LIEUTENANT WILLIAM N. SUTER, ASSISTANT-SURGEON.—Ordered to return to Washington Barracks, D. C., on the breaking up of the camp at Creedmoor, New York. S. O. 166, Division of the Atlantic, August 10, 1887.

COLONEL J. H. BAXTER, CHIEF MEDICAL PURVEYOR.—Ordered to proceed from Washington, D.C., to New York City, on public business, and on completion thereof to return to this city. Par. 5, S. O. 187, A. G. O., August 13, 1887.

LIEUTENANT-COLONEL JOSEPH C. BAILY, ASSISTANT MEDICAL PURVEYOR.—Granted leave of absence for one month. Par. 10, S. O. 191, A. G. O., August 18, 1887.

MAJOR W. D. WOLVERTON, SURGEON (Washington Barracks, D.C.).—Granted leave of absence for twenty days. Par. 3, S. O. 171, Division of the Atlantic, August 16, 1887.

MAJOR W. S. TREMAINE, SURGEON.—Found incapacitated for active service by an Army Retiring Board, and extension of leave of absence, on account of sickness, still further extended until further orders. Par. 9, S. O. 192, A. G. O., August 19, 1887.

MAJOR JOHN H. BARTHOLF, SURGEON.—Leave of absence extended one month. Par. 7, S. O. 196, A. G. O., August 24, 1887.

CAPTAIN JULIUS H. PATZKI, ASSISTANT-SURGEON.—Granted leave of absence for one month. Par. 15, S. O. 195, A. G. O., August 23, 1887.

CAPTAIN WASHINGTON MATTHEWS, ASSISTANT-SURGEON.—Ordered to proceed to Phœnix, Arizona Territory, on public business, and on completion thereof to return to his proper station, Surgeon-General's Office. Par. 21 S. O. 195, A. G. O., Aug. 23, 1887.

CAPTAIN CHARLES B. BYRNE, ASSISTANT-SURGEON (Washington Barracks, D. C.).—Granted leave of absence for one month, with permission to apply for an extension of one month. Par. 4, S. O. 171, Division of the Atlantic, August 16, 1887.

CAPTAIN BLAIR D. TAYLOR, ASSISTANT-SURGEON.—Granted leave of absence for twenty days, to take effect on or about August 31, 1887. Par. 7, S. O. 193, A. G. O., August 20, 1887.

CAPTAIN GEORGE F. WILSON, ASSISTANT-SURGEON.—Granted leave of absence for fifteen days. S. O. 78, Department of Dakota, August 8, 1887.

FIRST-LIEUTENANT WILLIAM D. DIETZ, ASSISTANT-SURGEON.—Granted leave of absence for two months, with permission to apply for an extension of one month. Par. 7, S. O. 189, A. G. O., August 16, 1887.

FIRST-LIEUTENANT W. D. MCCAW, ASSISTANT-SURGEON.—Relieved from temporary duty at Fort Riley, Kansas, and ordered to his proper station, Fort Leavenworth, Kansas. Par. 3, S. O. 84, Department of Missouri, August 15, 1887.

FIRST-LIEUTENANT E. L. SWIFT, ASSISTANT-SURGEON.—Ordered to report in person to the commanding general, Division of the Pacific, for duty with troops at Round Valley Indian Reservation. Par. 20, S. O. 195, August 23, 1887.

CAPTAIN HENRY JOHNSON, MEDICAL STOREKEEPER.—Ordered, in addition to his present duties, to take charge of the office and perform the duties of Acting Assistant-Medical-Purveyor in New York City, during the temporary absence on leave of Lieutenant-Colonel Joseph C. Baily, Assistant-Medical-Purveyor. Par. 11, S. O. 191, A. G. O., August 18, 1887.

OFFICIAL LIST OF CHANGES IN THE MEDICAL CORPS OF THE U.S. NAVY FOR THE WEEK ENDING AUGUST 20, 1887.

MEDICAL-INSPECTOR S. ROBINSON.—Placed on Retired List.

PASSED ASSISTANT-SURGEON J. E. GARDNER.—Detached from Naval Hospital, Norfolk, Virginia, and ordered to the Fish Commission Steamer "Albatross."

ASSISTANT-SURGEON WILLIAM MARTIN.—Ordered to Naval Hospital, Norfolk, Virginia.

SURGEON W. K. VAN REYPEN.—Appointed Medical Inspector from August 16, 1887.

PASSED ASSISTANT-SURGEON M. H. SIMONS.—Appointed Surgeon from August 16, 1887.

OFFICIAL LIST OF CHANGES OF STATIONS AND DUTIES OF MEDICAL OFFICERS OF THE U.S. MARINE HOSPITAL SERVICE FOR THE THREE WEEKS ENDING AUGUST 27, 1887.

LONG, W. H., SURGEON.—Leave extended six days, on account of sickness, August 13, 1887.

FESSENDEN, C. S. D., SURGEON.—Leave extended thirty days, on account of sickness, August 19, 1887.

GODFREY, JOHN, SURGEON.—Granted leave of absence for thirty days, August 17, 1887.

GLENNAN, A. H., PASSED ASSISTANT-SURGEON.—Granted leave of absence for thirty days, August 18, 1887.

MCINTOSH, W. P., ASSISTANT-SURGEON.—Granted leave of absence for twenty-five days, on account of sickness, August 17, 1887.

BAILHACHE, P. H., SURGEON.—Granted leave of absence for thirty days, August 26, 1887.

CARTER, H. R., PASSED ASSISTANT-SURGEON.—Granted leave of absence for twenty-seven days, August 25, 1887.

YEMANS, H. W., PASSED ASSISTANT-SURGEON.—Resignation accepted, to take effect September 30, 1887, and leave of absence extended to that date, August 24, 1887.

NORMAN, SEATON.—Granted leave of absence for six days, on account of sickness, August 27, 1887.